A DISSERTATION ON

"PROSPECTIVE STUDY ON EPIDEMIOLOGY, MANAGEMENT AND OUTCOME OF ACUTE INTESTINAL OBSTRUCTION IN ADULTS IN RGGGH"

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GENERAL SURGERY

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THE TAMILNADU Dr.MGR MEDICAL UNIVERSITY

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CERTIFICATE

This is to certify that, the dissertation entitled **"PROSPECTIVE STUDY ON EPIDEMIOLOGY, MANAGEMENT AND OUTCOME OF ACUTE INTESTINAL OBSTRUCTION IN ADULTS IN RGGGH"** is the bonafide work done by **Dr.MOHANRAJA.P,** during his MS (General Surgery) course 2010-2013, done under my supervision and is submitted in partial fulfillment of the requirement for the M.S.(BRANCH-I)- General Surgery of The Tamilnadu Dr.MGR Medical University,April 2013 examination.

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DECLARATION

I, **Dr.MOHANRAJA.P**, certainly declare that this dissertation titled **"PROSPECTIVE STUDY ON EPIDEMIOLOGY, MANAGEMENT AND OUTCOME OF ACUTE INTESTINAL OBSTRUCTION IN ADULTS IN RGGGH"** represents a genuine work of mine. The contributions of any supervisors to the research are consistent with normal supervisory practice, and are acknowledged.

I also affirm that this bonafide work or part of this work was not submitted by me or any others for any award, degree or diploma to any other University board, either in India or abroad. This is submitted to The TamilNadu Dr. M.G.R Medical University, Chennai in partial fulfillment of the rules and regulations for the award of Master of Surgery Degree Branch I (General Surgery).

Date:

Place: **Dr.MOHANRAJA.P**

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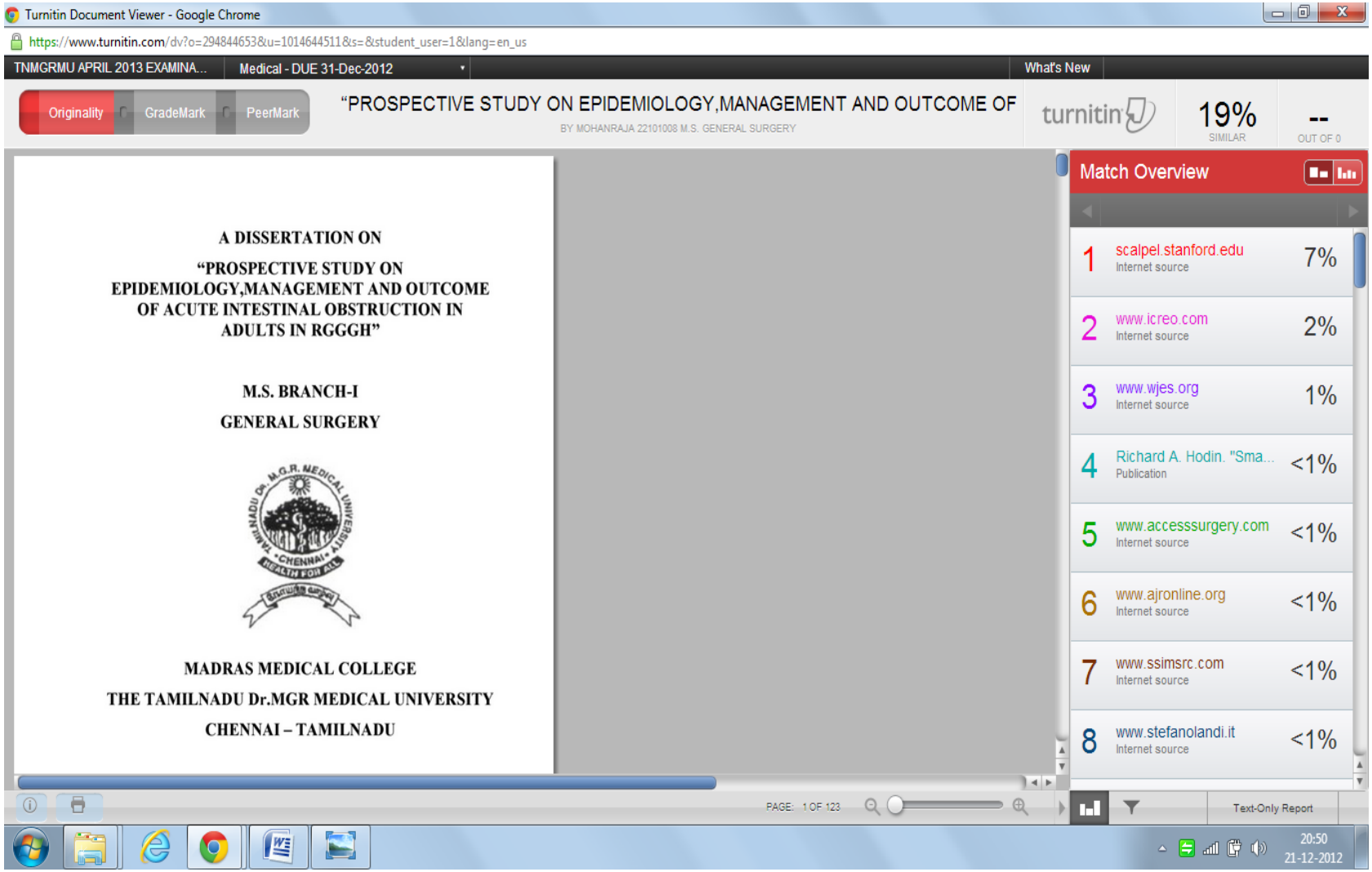
Dr.MOHANRAJA.P

CONTENTS

LIST OF ABBREVIATIONS

AIO	-	Acute intestinal obstruction
BP	-	Blood pressure
CT	-	Computed tomography
CVP	-	Central venous pressure
ECF	-	Extracellular fluid
ECG	-	Electrocardiography
ESR	-	Erythrocyte sedimentation rate
GI	-	Gastrointestinal
GIT	-	Gastrointestinal tract
IMA	-	Inferior mesenteric artery
IV	-	Intravenous
IVC	-	Inferior venacava
KCl	-	Potassium chloride
PVP	-	Polyvinyl propylene
RBC	-	Red blood cells
RIF	-	Right iliac fossa
RR	-	Respiratory rate
S	-	Sacral
SMA	-	Superior mesenteric artery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INTRODUCTION

Bowel obstruction remains one of the most common intra-abdominal problems faced by general surgeons in their practice whether caused by hernia, neoplasm, adhesions or related to biochemical disturbances intestinal obstruction of either the small or large bowel continues to be a major cause of morbidity and mortality.[1] They account for 12% to 16% of surgical admissions for acute abdominal complaints. Manifestations of acute intestinal obstruction can range from a fairly good appearance with only slight abdominal discomfort and distension to a state of hypovolemic or septic shock (or both) requiring an emergency operation.

To identify and analyse the clinical presentation, management and outcome of patents with acute mechanical , obstruction along with the etiology of obstruction and the incidence and causes of bowel ischaemia, necrosis and perforation.[2]

The death due to acute intestinal obstruction is decreasing with better understanding of pathophysiology, improvement in diagnostic techniques, fluid and electrolyte. Early diagnosis of obstruction skillful operative management, proper technique during surgery and intensive postoperative treatment carries a grateful result.

AIMS AND OBJECTIVES

1. To study the various modes of presentation, various Causes, importance, Early recognition, diagnosis, management.

2. To study the influence of various factors like, age, sex, diet, socio- economic status in the pathogenesis of acute intestinal Obstruction in adults.

3. To study the morbidity and mortality rates in acute intestinal Obstruction

REVIEW OF LITERATURE

HISTORICAL REVIEW

The attempts to treat acute intestinal obstruction dates back to centuries. In 6th century Sushrutha wrote oldest known descriptions of bowel surgery. Forms of intestinal obstruction like strangulated hernia, intussusception were known to the ancient Egyptians. Intestinal obstruction was observed by Hippocrates (460-370 BC). The earliest operation recorded was performed by Proxogorous (350 BC), who created enterocutaneous fistula to relieve obstruction.

- Fabricus d'Aquopendente in 12th century described a procedure of intestinal repair involving end-to-end anastamosis

- John Arderence (1306-1390) was the first surgeon who wrote the book on "Passio Iliaca" (Appendicitis or intestinal obstruction).

- Ambrosis Pare (1510-1590) was first to recognize obstruction as a pathological entity. For severe cases he used mercury in water, lead bullets smeared with mercury[3].

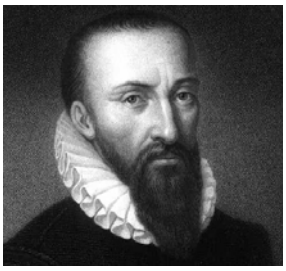


Figure 1: Ambrosis Pare

- Franco (1561) did first Surgery on strangulated hernia.
- Kerckring in 1670 described the intestinal valvulae conniventes.
- Bonetos in 1679 treated intussusception surgically.
- Amsterdam in 1676 suggested opening of abdomen to treat intussusception and volvulus.
- Mery in 1701, removed several feet of gangrenous bowel and established an artificial anus in a woman suffering from strangulated hernia.
- La Peyronie in 1723, reported having where he excised the devitalized bowel and creating an artificial anus and mucous fistula, which was closed later on.
- Mensching in 1756 used repeated intestinal puncture to treat obstructed bowels.

- Planned Caecostomy was first described by Pillore (1776).
- Duret performed the first successful sigmoidostomy for the relief of obstruction in1793.
- Dupytren in 1800 established enterostomy for obstruction.
- In 1826, Lipperance resected the lower rectum and anus and created colostomy, and in 1836, Diffenbach did resection anastomosis of small bowel in strangulated hernia.
- Duchenne in 1855 reported several successful instances where he treated intestinal obstruction with faradic current.
- Volvulus of caecum was first described by Rokintansky in 1841.
- In 1875 Elliott performed bowel resection for mesentric vascular occlusion exteriorly.
- In 1880 Block Parker and Kerr safely did exteriorisation, primary resection and anastamosis in different cases.
- Roentgen in 1893 discovered X-ray and Schwartz in 1911 pointed out the virtue of scout fIlm; Kloiber of Germany (1919) emphasized the importance of X-ray in locating the level of obstruction.[4]

- How toxic substances get accumulated bowel was described by V/hipple and Williams (1926) described influence of anaerobic infection as a cause of toxaemia.

- Einborn (1909) passed a 18° F tube to study small bowel contents and in 1926 vander Reir and Schamber passed a tube through whole of GIT.

- In 1925, Gamble et al. proved that loss of fluid and solutes from the body into the bowel lumen was the factor responsible for death.

- Oppenheimer and Mann (1934) studied microcirculation, caecostomy was first described by Allen and Welch in 1947. Noer et al in 1951 confirmed circulatory disturbances brought about by increased distension.

- Johnston in 1938, Harris in 1945, Cantor in 1946 and Gafton Smith in 1952 described various other tubes for gastrointestinal drainage.

- Bishop and Allock (1960) studied the bacteriology of gut above the obstruction.

- The experimental use of Doppler ultrasonography to determine the viability of ischaemic intestine was first described by Wright and Gobson in 1975.

- Marfuggi and Greenspan in 1981 reported 93% accuracy of fluoroscopic dye injection technique for determining the viability of inschaemic bowel.

- Bookstein in 1982, used angiography to diagnose and treat small bowel bleeding.

- In 1996, Akgun gained more attention in mesosigmoplasty as definitive operation sigmoid volvulus.[5]

- In 1997, Yaco et al. evaluated diagnostic procedure, for diverticular disease (CT scan, contrast enema, ultrasonography).

EMBRYOLOGY OF SMALL INTESTINE

During the early stage of development, the primitive gut is in free communication with the rest of the yolk sac. In the cephalic and caudal parts of the embryo the primitive gut forms a blind ending tube the foregut and the hindgut and the middle part, the midgut remains temporarily connoted to the yolk sac.

In the 5th week embryo, there will be rapid elongation of the gut and its mesentery resulting in formation of the primary intestinal loop. The cephalic limb of the loop develops into the distal part of the duodenum the jejunum and part of the ileum. The caudal limb becomes the lower portion of the ileum, the caecum, the appendix, the ascending colon and the proximal two-thirds of the transverse colon. The hindgut gives raise to distal third of transverse colon, the sigmoid, the rectum and part of anal canal.

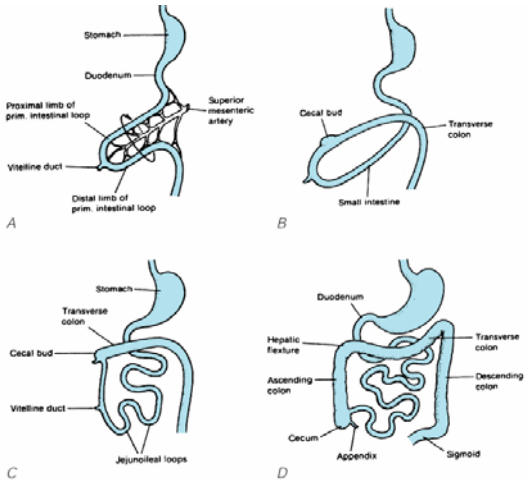


Figure 2: Developmental rotation of the intestine

Chronology of Rotation of the Midgut Loop

The loop has a prearterial or proximal segment and post arterial or distal segment. Viewed from the ventral side, the loop undergoes an anticlockwise rotation by 90°, so that it now lies in the horizontal plane.

The pre-arterial segment comes to lie on the right side and the post-arterial segment on the left.

- Pre-arterial segment now undergoes great increase in length to form coils of the jejunum and ileum and the loops still lie outside the abdominal cavity to the right side of the distal limb.

The coils of the jejunum and ileum (pre-arterial) return to the abdominal cavity.The coils of jejunum and ileum pass behind the superior mesenteric artery into the left half of the abdominal cavity.

- The post-arterial segment of the midgut loop returns to the abdominal cavity, it also rotates in an anticlockwise direction so that the transverse colon lies anterior to the superior mesenteric artery and the caecum comes lie on the right side.

- Gradually the caecum descends to the iliac fossa and the ascending, transverse and descending parts of the colon become distinct.

There are three errors in the stages of rotation.

1. Non-rotation

2. Reversed rotation

3. Malrotation

Pathological consequences of anomalies of rotation[6]

- No functional disturbance may result from abnormal fixation.
- Deficient fixation causes ptosis, torsion and volvulus.
- Excessive fixation may cause interference with mobility, kinks and compression of intestine.
- Abnormal rotation predisposes to volvulus, which causes intestinal obstruction.
- Volvulus of the ileocaecal segment is the typical lesion in later life resulting from imperfect rotation or deficient fixation of the gut.

Accessory bands of peritoneum

can cause (1) Intestinal obstruction (2) Kinking (3) Angulation of bowel.

Failure of part of the original membrane to disappear, minor alterations in the development of secondary mesentery result in accessory peritoneal bands.

These are:

- **Lane's ileal band:** The thickened peritoneal band extending from the right iliac fossa to the 5 cm of ileum which on continuous contraction causes kinking of the small bowel and resulting in obstruction.

- **Mesosigmoid membrane** (Lane's first and last band): This is formed by the thickening of peritoneum extending from the pelvic brim of left iliac region to the junction of descending and sigmoid colon.

- **Genitomesenteric fold of Douglas:** causes kinking of appendix causing obstructive appendicitis as it extends from the back of the terminal mesentery to the region of the suspensory ligament or ovary or testis.

- **Jackson's membrane:** Lies between the posterior abdominal wall and caecum or ascending colon on the right side or from the hepatic flexure to caecum.

ANATOMY

Small intestine

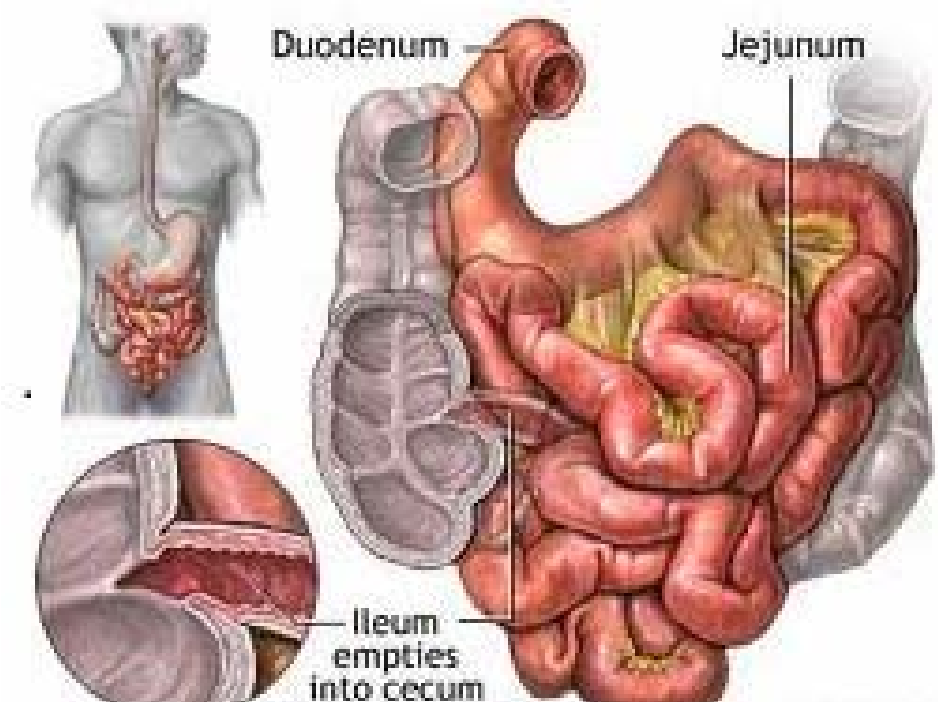


Figure 3: Small intestine

The small intestine is the longest part of the gastrointestinal tract and extends from the pyloric orifice of the stomach to the ileocaecal fold. This hollow tube, which is approximately 6-7 m long with a narrowing diameter from beginning to end, consists of the duodenum, the jejunum and the ileum.[7]

The adult duodenum is 20-25 cm length and the name coined as duodenum because length is as long as width of 12 fingers. It is shortest,

widest and most fixed part. It has no mesentery and partially covered by peritoneum. Its course presents a remarkable curves somewhat like horseshoe type, the convexity being directed towards the right and concavity to the left embracing the head of the pancreas. It has been divided into four portions. First part (superior portion), Second part (descendingportion), third part (horizontal portion) and fourth part (ascending portion).

Blood supply and nerve supply

Arteries supplying the duodenum arise from the right gastric, supraduodenal, right gastroepiploic, and superior and inferior pancreaticoduodenal arteries.

Veins: These end in the splenic, superior mesenteric and portal veins.

Nerves: They come from the coeliac plexus.

Lymph nodes: Along interior and posterior pancreatico duodenal artery.

Jejunum and ileum

In small intestine excluding duodenum, upper 2/5 are formed by jejunum and lower 3/5 are ileum. The rest of the small intestine extends from the duodenojejunal flexure to the ileocaecal valve, ending at the

junction of the caecum and ascending colon. It is totally covered by peritoneum and it is arranged in a series of coils attached to the posterior abdominal wall by the mesentery.

The jejunal loops characteristically situated in the upper abdomen to the left of midline, whereas the ileal loops tend to lie in the lower right part of the abdomen and pelvis. This distribution can be reversed during paralytic ileus or small bowel obstruction due to rotation around the mesenteric attachment following bowel distension.

Blood supply

Blood supply is by superior mesenteric artery which is a branch of aorta, thebranches of which, reaching the mesenteric border, extend between the serosal and muscular layers. After this, numerous branches traverse the muscle, supplying it and forming an intricate submucosal plexus from which minute vessels pass to glands and villi. The superior mesenteric veins follow the arteries.

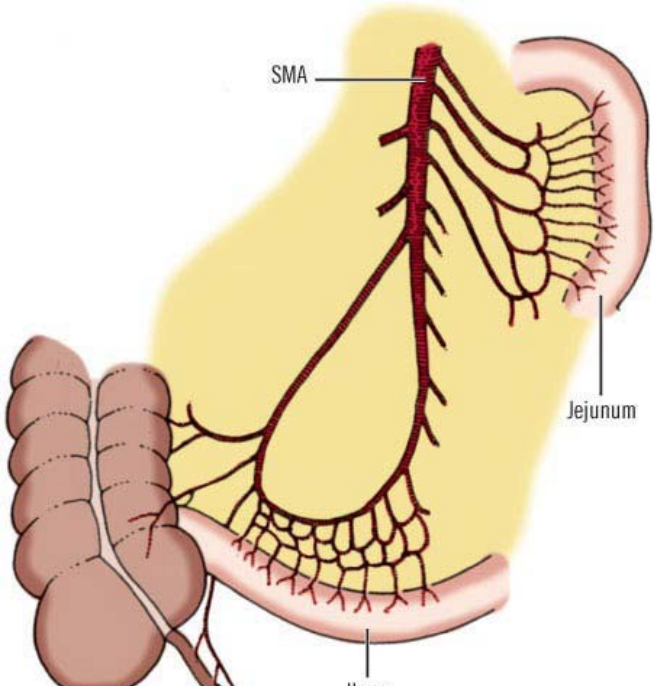


Figure 4: Blood supply of small intestine

Nerve supply

Nerve supply by vagi and thoracic splanchnic nerves through the celiac ganglia and superior mesenteric plexus.

Large intestine

It is about 150 cm long, it extends from the terminal ileum to the anus. Its function is chiefly absorption of fluids and solutes and it differs in structure, size and arrangement from the small intestine in the following ways:

- It is for the most part more fixed in position.
- Its longitudinal muscle, though a complete layer, is concentrated into three longitudinal taeniae coli.
- The colonic wall is puckered into sacculations (haustrations) and appendices epiploicie by the taeniae.
- It has a great caliber.

The divisions are caecum, colon proper and the rectum.

Caecum

The caecum is a blind pouch lies in the right iliac fossa, its average axial length is about 6 cm and its breadth about 7.5 cm continues proximally with the distal ileum and distally with the ascending colon and is related posteriorly to iliopsoas muscle and femoral nerve, anteriorly to the abdominal wall, greater omentum and the coils of ileum. Almost the entire posterior surface of caecum is attached to the abdominal wall, in some cases it is wholly unattached.

Ileocaecal valve

The ileum opens on its posteromedial aspects of the caeco-colic junction and two flaps which project into the lumen of the colon. The

valve is actually closed by sympathetic tone. It is mechanically closed by the distensions of caecum and prevents the reflux of caecal contents into the ileum and regulates the flow of ileal contents.

Colon

The colon is conveniently considered in four parts: (1) Ascending, (2) Transverse, (3) Descending and (4) Sigmoid.

Blood supply

Blood supply is by branches of superior mesenteric artery and inferior mesenteric artery. Superior mesenteric artery supply upto the junction of middle 1/3rd of transverse colon and colon beyond this is supplied by inferior mesenteric artery.

Nerve supply

Sympathetic to midgut from coeliac ganglion (T1-L1). Parasympathetic from vagus through coeliac plexus. Hindgut portion receives sympathetic supply from the lumbar sympathetic chain from L1-L2 and parasympathetic from the pelvic splanchnic nerves.

Rectum

The rectum is 12 cm long and is continuous with the sigmoid colon at S3. Thehuman rectum follows the posterior concavity of the sacrum and shows three lateral curves or flexures that are most prominent when the viscus distended, upper and lower curves convex to the right and a middle curve convex to the left, the lowest part is slightly dilate as the rectal ampulla. It ends 2-3 cm in front and below the tip of the coccyx, turning abruptly downwards and backwards through levator ani muscle to become the anal canal 4 cm from the anal verge. The mucosa of large intestine differs from small intestine in that it has no villi and consists of simple columnar epithelium which has only absorptive and goblet cells. The longitudinal muscles of muscularis mucosa are thickened to form taeniacoli, on contraction leads to formation of hanstra necessary for haustral churning.

Blood supply

Blood supply mainly from the superior rectal artery, with contributions from the middle and inferior rectal and median sacral vessels. Veins correspond to the arteries, but anastomose freely with one another, forming an internal rectal plexus in the submucosa and external rectal plexus outside the muscular wall.

Nerve supply

The sympathetic is derived by branches from the hypogastric plexus. The parasympathetic supply is from S2 and S3 by the pelvic splanchnic nerves.

Lymphatic drainage of colon

Lymph from the colon passes through four sets of lymph nodes: (a) Epicolic lymph nodes, lying on the wall of the colon, (b) Paracolic nodes on the medial side of ascending, descending and mesocolic border of transverse and sigmoid colon, (c) Intermediate nodes along the main branches of vessels, (d) Terminal nodes at the origin of SMA and IMA, finally drains to para-aortic nodes.

CLASSIFICATION OF INTESTINAL OBSTRUCTION

Acute intestinal obstruction is most commonly a surgical disorder of small intestine and accounts for approximately 20% of all surgical admissions.

Intestinal obstruction may be classified into two types.

1. Dynamic obstruction

2. Adynamic obstruction

DYNAMIC OBSTRUCTION: Where peristalsis working against a mechanical obstruction.

Irrespective of aetiology or acuteness of onset, in dynamic obstruction the proximal bowel dilates and develops an altered motility. Below the obstruction the bowel exhibits normal peristalsis and absorption until it becomes empty at which point it contracts and becomes immobile.[9]

The causes of intestinal obstruction are:[10]

Intraluminal

- Intussusception
- Bezoar
- Foreign bodies
- Gallstones
- Mucosal tumours

Intramural

- Stricture
- Malignancy: Carcinoid, Lymphoma, Leiomyosarcoma
- Inflammation: Crohn’s disease, Tuberculosis
- Haematoma
- Endometriosis

Extramural

- Bands/adhesions
- Hernia: External – Inguinal, Femoral, Incisional, Obturator
  Internal – Paraduodenal, Epiploic foramen,
  Diaphragmatic,Transmesenteric
- Tumours: Peritoneal metastasis, Desmoid
- Abscess: Diverticulitis, Pelvis inflammatory disease, Crohn's disease

ADYNAMIC OBSTRUCTION: This may occur in two forms:

a. Peristalsis may be absent e.g.: Paralytic ileus

b. Peristalsis may be present in a non-propulsive form e.g.: (1) Mesenteric vascular occlusion (2) Pseudoobstruction.

CLINICAL FEATURES

Cardinal features of intestinal obstruction are:

1. Pain abdomen
2. Vomiting
3. Distension of abdomen
4. Constipation

1. Pain abdomen

Abdominal pain is the first symptom. The onset may be insidious or abrupt in simple obstruction, but with strangulation the onset is usually sudden and severe. The pain is diffuse, poorly localized and is felt across

the upper abdomen in high obstruction, at the level of the umbilicus in low ileal obstruction, in the lower abdomen in colonic obstruction and in the perineum as well as in rectosigmoid obstruction.

In high intestinal obstruction (the period between attacks of pain is short 3-5 minutes), where as it is longer in low obstruction (15 to 20 minutes). Recurrent attacks of severe abdominal pain is a leading feature of all varieties of acute intestinal obstruction with the sole exception of paralytic ileus.

2. Vomiting

Vomiting is the next most common symptom. A constant symptom, the early vomiting is reflex in nature followed by quiescent period before real vomiting due to obstruction resumes. This quiescent period is of shorter duration in high-level obstruction and longer in lower small bowel obstruction. As acute intestinal obstruction progresses, the character of the vomitus alters. Initially it contains partly digested food, followed by bilious vomiting. Finally it is faeculent.

In high-level obstruction vomiting is frequent, copious and projectile, in lowlevel small bowel obstruction vomiting is less projectile and less frequent. Vomiting is unusual in colonic obstruction. Reflex

vomiting is unusual in colonic obstruction because the ileocaecal valve is competent. Colonic obstruction is associated with the brown vomit which becomes increasingly foul smelling as the obstruction persists.[11]

3. Distension

In early cases of obstruction of the small intestine abdominal distension is often slight or even absent. When the proximal jejunum is obstructed, the stomach becomes distended with gas and accumulated secretion, so that the epigastric region may, in later stages be more prominent and tense. When the ileum is involved, the central portion of the abdomen is moderately blown out and when the distal colon is blocked, there is considerable universal distension of abdomen, with well-marked bulging in the flanks. Visible peristalsis may be present.

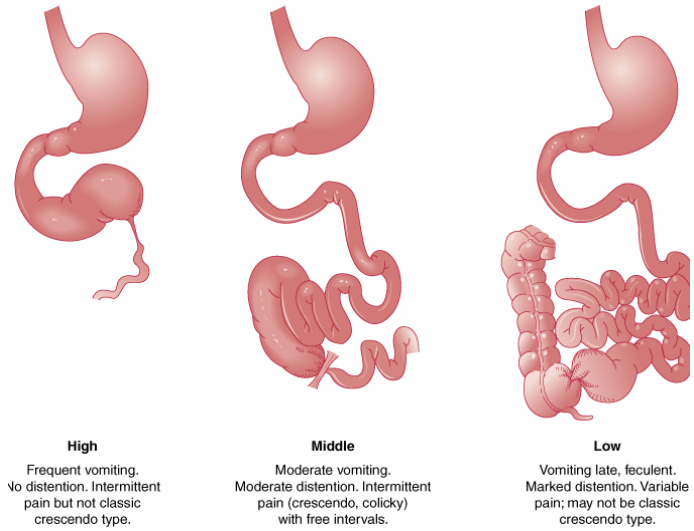


Figure 5: Clinical features of intestinal obstruction

4. Constipation

In complete intestinal obstruction, after the contents of the bowel below the obstruction have been evacuated, there is constipation and usually neither faeces nor flatus is passed i.e. absolute constipation.

The rule of constipation is present in intestinal obstruction does not apply in cases of Ritcher's hernia, gall stone obstruction, mesenteric vascular occlusion.

History and physical examination

A detailed history and physical examination helps in diagnosis and management of intestinal obstruction. In simple mechanical obstruction there will be very few abdominal signs. Whereas in strangulated obstruction, patient will be toxic, tachycardia and hypotension will be there. Any past history of abdominal surgery, acute cholecystitis, appendicitis or any other intraabdominal infections, suggests adhesion as a cause of obstruction. Hernia of long duration gives rise to strangulation, there may be one of the following history.

- Alternate diarrhoea and constipation with loss of weight suggests tuberculosis and malignancies.

- Recent onset of constipation suggests malignancy in elderly people.

Physical examination

Skin turgor: may be lost due to dehydration, may be cold and calmy.

Tongue: which may be dry and coated due to dehydration.

Nail and sclera: Anaemia, jaundice may be evident.

Rapid low volume pulse, low blood pressure, cold extremities, anxious look and increased respiratory rate are the evidence of shock and septicaemia.

Examination of abdomen

- **Inspection:** On inspection previous surgical scars which indicates adhesions or cancer. In early stage visible peristalsis may be seen. All hernial orifices has to be looked.

- **Type of abdominal distension:** Central in small bowel obstruction and upperabdominal in high-level obstruction. Distension will be more in the flanks in colonic obstruction.

- **Type of peristalsis:** Central step ladder type of peristalsis seen in distal small bowel obstruction and right to left over the umbilicus in the colonic obstruction.

Palpation

- Abdomen must be examined for presence of any palpable mass, localized abdominal tenderness, rebound tenderness and mass is suggestive of strangulation. In peritonitis there will be generalized rigidity and tenderness. During pain heaped up coils of intestine or prominent distended coils may be seen.

- **Auscultation:** In simple mechanical obstruction, sounds become loud, high pitched and metallic. In late stages bowel sounds may be absent due to paralysis of bowel musculature. Bowel sounds may be absent in strangulation and ileus or low-pitched tingling sounds may be heard due to movements of fluid from one coil to another.

- **Rectal examination:** To be performed in all cases of obstruction, may reveal faecal impaction, mass, red current bleeding in intussusception. A palpable pelvic mass or bulge due to collection in the Pouch of Douglas may be present.

Ballooning of the rectum usually occurs in the intestinal obstruction may be due to obstruction to nerves causing sympathetic paralysis.

CLINICAL FINDINGS IN SMALL BOWEL OBSTRUCTION

Features	**Proximal/High obstruction**	**Distal/low obstruction**
Onset of symptoms	Sudden	Gradual
Pain	Epigastric, intense, colicky usually relieved by vomiting	Periumbilical, colicky
Vomiting	Early, bilious, voluminous, frequent	Later, infrequent feculent
Tenderness	Epigastric or periumbilical mild unless strangulated	Diffuse and progressive
Distension	Absent	Diffuse and progressive
Obstipation	Absent or mild	Mild to moderate
Radiologic findings	Distended proximal small bowel loops or gasless	Diffusely distended small bowel loops, air fluid levels

LABORATORY INVESTIGATIONS

Diagnostic aspiration

So important in the distinction between simple and strangulated obstruction, aspiration of peritoneal cavity with a fine needle in case of doubt and withdraw of blood stained fluid is diagnostic. of strangulation. Root finds that significant increase in the polymorphonuclear count of

peritoneal fluid can be detected within three hours after peritoneal invasion.

X-ray diagnosis

In all cases, ‘Scout’ film of the abdomen, with the patient standing and lying false fluid levels. The sensitivity of abdominal radiographs in the detection of small bowel obstruction ranges from 70 to 80%.[12]

The finding in erect abdomen X-ray for small bowel obstruction is the triad of dilated small down must be taken. No enema should be given prior to plain X-ray because it bowel loops (>3 cm in diameter) air fluid levels seen on upright films and a paucity of air in the colon.[13]

Gas shadows: When the jejunum, ileum or the colon is distended with gas, each structure has significant radiological pictures. Jejunum is characterised by ‘valvulae conniventes’ that pass from anti-mesenteric may cause to mesenteric border in the regular fashion. Ileum radiography was described by Wangensteen as being ‘characterless’. Large intestine shows haustral markings which unlike the valvulae conniventes are spaced irregularly, do not completely traverse the circumference of the bowel and gas shadows of large intestine are located peripherally.

Fluid levels: In adults, two inconstant fluid levels are regarded as physiological, one at the duodenal cap and the other within the terminal ileum. In obstruction, fluid levels appear later than the gas shadows; nearer the obstruction the ileocaecal valve fluid level is proportional to the degree of obstruction and to its site in small intestine.

In upper small bowel obstruction, fluid levels will be in left upper quadrant and there will be few in number, multiple fluid level seen all over the abdomen in case of low small bowel obstruction. The presence of gas in the wall of the bowel is highly significant sign of intestinal necrosis, which was demonstrated by Schorr in 1963.

Volvulus of sigmoid shows greatly distended sigmoid loop filling the whole of the abdomen upto the diaphragm with the “Bent inner tube sign”. Millin and Righler pointed out that “coffee bean” sign in pathognomonic sign of caecal volvulus.

(a) Crohn’s disease: String sign – narrow and smooth terminal part of ileum is seen.

(b) Hyperplastic ileocaecal tuberculosis: A long narrow constricted terminal ileum and ascending colon with caecum can be seen.

Barium enema: In intussusception, barium is seen as a ‘claw’ around a negative shadow of intussusception whereas in sigmoid volvulus, barium column ends at the level of the distal sigmoid torsion in a characteristic Twisted Bird’s Beak deformity.

Computerized tomography (CT)

CT demonstrates the cause of obstruction. Computerized tomography is very much useful in revealing the site, level and cause of obstruction and in displaying signs of threatened bowel viability.

CT is most valuable when there are systemic signs suggestive of infarction, an associated palpable mass. In these cases CT may confirm the presumptive diagnosis or reveal other causes such as appendicitis or diverticulitis.

It is a procedure of choice in patient who have a history of abdominal malignancy and clinical symptoms suggestive of bowel obstruction. In strangulated obstruction, target sign or pneumatosis and haemorrhage in the mesentery can be seen.

TREATMENT OF ACUTE INTESTINAL OBSTRUCTION

With some exceptions, an urgent surgical intervention is needed in a case of intestinal obstruction. Although it is difficult to differentiate

between a simple and strangulated obstruction, the assessment of patient is done by taking detail history and clinical examination. Investigations are to find out whether the obstruction is mechanical or a dynamic and the level of obstruction.

The treatment has to be planned accordingly to the above assessment which includes supportive management and surgical management. There are four main measures in management of obstruction.

- GI decompression
- Fluid and electrolyte replacement
- Relief of obstruction usually surgical
- Antibiotics to prevent complications from associated sepsis

The first two steps are always necessary prior to surgical relief of obstruction and are main stay of post-operative treatment. Surgical treatment is necessary for most cases of intestinal obstruction, but should be· delayed until resuscitation is complete provided there are no sign of strangulation or evidence of closed loop obstruction.

Conservative management

Patients with a partial intestinal obstruction may be treated conservatively with resuscitation and tube decompression alone.

Resolution of symptoms and discharge without the need for surgery have been reported in 60-85% of patients with a partial obstruction.[14]

Simple obstruction caused by postoperative early adhesions or kinking may resolve spontaneously with conservative management and it is indicated in following conditions.

- Postoperative early adhesions
- Paralytic ileus of non-paralytic origin
- Inflammatory condition causing obstruction
- Obstruction due to worm impaction

The initial conservative management is used in above said conditions, facilitates spontaneous relief of obstruction and avoids more of adhesion formation to surgery. The decision to intervene depends largely on the underlying cause, clinician involved and general condition of the patient. The conservative management includes.

- GI decompression
- Fluid and electrolyte replacement
- Antibiotics to prevent complications

GI decompression

There are two types of aspiration tubes used in GI decompression and the decompression is achieved by the passage of non-vented (Ryle's tube) or vented Salem tube which are normally placed on free drainage with hourly aspiration but may be placed on continuous or intermittent suction decompression of the bowel proximal to obstruction and stomach will relieve certain amount of distension and toxic fluid accumulated in the bowel. Also avoids the aspiration pneumonia during induction of anaesthesia. It is also improves local bowel circulation and venous return to the heart by relieving the pressure over the IVC. There are other special tubes used in decompression of the small bowel (long intestinal tube).

Fluid and electrolyte replacement

The individual patient has to be assessed according to general condition of the patient and underlying cause of obstruction. This should be routine in all cases of bowel obstruction before taking up for surgical intervention except in few case of early simple obstruction, which is within 24 hours. The longer the duration of obstruction longer will be the time taken to get the patient ready for surgery. It is best to intervene when vital signs show a return to normal. It is unwise to operate early in a case

of prolonged obstruction with poor general condition and it is also not wise to delay in case of strangulated and close loop obstruction.

The parameters like pulse rate, BP, Shock state, Degree of dehydration, Urine Output, Initial haematocrit value are taken into consideration in fluid management.

Antibiotics

Use of broad-spectrum antibiotics in adequate doses along with metranidazole are advised. It is important to give antibiotics pre and postoperatively till adequate recovery takes place.

Surgical Management

With regard to the timing of surgery, all patients should be operated onpromptly after volume resuscitation if any evidence or suspicion araises that bowel is ischaemic.[15] Early operation indicated in (1) obstructed and strangulated hernia, (2) internal intestinal strangulation (3) acute obstruction, The classical clinical saying that the sun should not set and rise in case of unrelieved intestinal obstruction is sound and should be followed.

Laparotomy

In patients with small bowel obstruction who have not had previous abdominal surgery or in those with clinical evidence of ischaemia, a laparotomy is mandatory.[16]

When the cause of obstruction lies within the abdomen and but its site is doubtful, right paramedian incision is advised, if left sided colonic obstruction is defined left mid or lower paramedian incision preferred.Haemorrhagic fluid denotes strangulation; clear straw-coloured fluid denotes simple obstruction. The operative assessment is directed to:

- Site of obstruction
- Viability of the gut
- The nature of obstruction

The type of surgical procedure required will depend upon the nature of the cause, following relief of obstruction the viability of the involved bowel should be carefully assessed. In case of viable bowel, peritoneum will be shiny, mesentery bleeds on prick whereas nonviable bowel, peritoneum is lusterless, mesentery does not bleed on prick.

Difference between viable and non-viable bowel

Intestine	Viable	Non-viable
Circulation	Dark colour become lighter, mesentery bleed on prick	Dark colour remains mesentery does not bleed on prick
Peritoneum	Shiny	Dull and lusterless
Intestinal musculature	Firm, Pressure rings may or may not disappear, peristaltic movements may be observed	Flabby, thin and friable pressure rings persist, no peristaltic movements observed

Doppler sonography can also help to test the circulation in the mesenteric vasculature (a most accurate method of testing viability).

If viability of the bowel is in doubt, it should be dropped in warm moist pads for ten minutes along with 100% oxygen in the anaesthetic gas. Then after ten minutes it has to be reassessed, in doubtful cases resection has to be done.

Principles of large bowel obstruction

As most of the large bowel obstruction are due to malignancy, volvulus or secondary to adhesive bands, which commonly occur, in elderly patient.

When lesion is operable and found in the caecum, ascending colon or proximal transverse colon, an emergency right hemicolectomy should be performed; if lesion is fixed a proximal stoma (colostomy. or ileostomy if ileocaecal wall is incompetent) or an ileo transverse colon bypass has to be considered, whereas obstruction lesions, of splenic flexure (malignant) should be treated by an extended right hemicolectomy. If one stage resection anastomosis infeasible a covering colostomy to protect the site of anastomosis is safe, where the distal segment could not be brought to the surface a proximal stoma and the distal end closed and returned to abdomen (Hartman's procedure) or both the ends brought outside, proximal as stoma and distal as mucus fistula, followed by second stage colorectal anastomosis can be planned when patient is fit. In very old or enfeebled patients when an obstructing carcinoma of rectum is fixed, left iliac colostomy is the best site for a permanent artificial anus. In rare circumstances, or if caecal perforation is suspected, wait for some time for improving the patient's condition and later relieving of obstruction can be done by doing an emergency caecostomy through a small incision in right iliac fossa.

INTESTINAL OBSTRUCTION BY ADHESIONS AND BAND

It is the most common cause of intestinal obstruction in developing countries. The pathology lies with peritoneal irritation results in local outpouring of fibrin which produces adhesions between opposed peritoneal surfaces. The fibrinous adhesions may become vascularised and become mature fibrous tissue. Infection being an important cause. Also foreign materials like silk thread, barium sulphate, talc, results in fibrous formation.

These commonly occurs following laparotomy surgeries. Once adhesions have developed, progression to obstruction is inevitable in a significant proportion.[17] Ileum is the commonest segment to be obstructed due to adhesions. After abdominal surgeries, about 5% of the cases subsequently develop obstruction due to adhesions, whereas operation in the colon carry high incidence of obstruction, about 1/5th of cases. About 20% of the obstruction occurs in the first year after laparotomy and most of these occur during first few weeks after surgery and termed as early postoperative obstructions and most resolved by conservative treatment.[18]

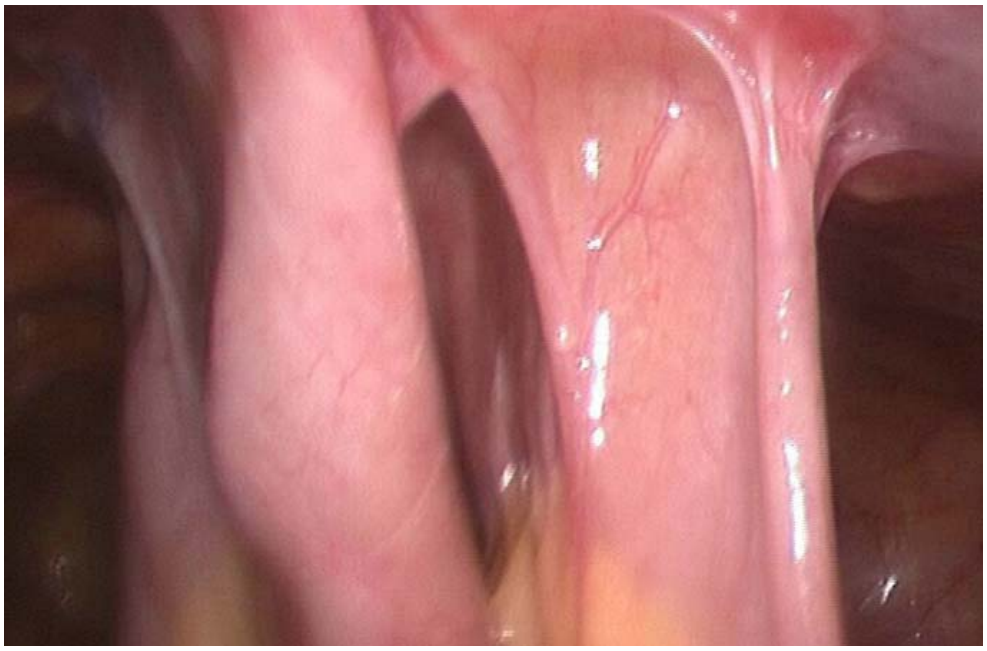


Figure 6: Postoperative adhesions

Type 1: Postoperative fibrinous adhesions cause incomplete obstruction. This occurs between 3-6th postoperative day. Also called as bread and butter fibrinous adhesions. These types of adhesions are managed conservatively.

Type 2: Strong bands of postoperative fibrous adhesions and occur at an organ where there is deficient blood supply and takes additional blood supply through these adhesions, and these may cause intestinal obstruction at any time after abdominal operations.

Type 3: Adhesion of loops of intestine to an inflamed intraperitoneal structure, e.g. Tubercular mesenteric lymphadenitis, Salpingitis.

Type 4: This follows chemical irritation from material such as talc glove powder or powdered antibiotics placed during laparotomy.

Prevention for adhesion[19,20]

The following factors may limit adhesions formation:

- A good surgical technique.
- Minimal contact with gauze.
- Washing of the peritoneal cavity with saline to remove clots and debris.
- Covering the anastamotic and raw peritoneal surface with omentum.
- Many substances have been instilled in the peritoneal cavity to prevent adhesion formation like hyalurinidase, hydrocortisone, silicon, dextron, polylyvinyl propylene (PVP) chondritin, streptopyris and anticoagulants, antihistamine, NSAIDs, streptokinase. But no single agent found to be safe and effective.

Treatment

The treatment for adhesion is same as the general principles of management often it is curative in early type of adhesions, but conservative treatment should not prolong beyond 48-72 hours and should not be continue if symptoms and signs are progressive even after

initial resuscitation. When laparotomy is done multiple adhesinolysis can be done.

Recurrent intestinal obstruction due to adhesion

Adhesions are a major cause of late morbidity.[21] Approximately 10% of all patients who have laparotomy for adhesive obstruction will require a further operation at a later date for the same problem. A further 10% may require a third operation for adhesive obstruction.

There are often chances of recurrent obstruction after first adhesinolysis. The following procedures may be considered for recurrent obstruction

- Intestinal intubation
- Charles Phillips transmesenteric plication
- Noble plication
- Repeat adhesionlysis (enterolysis)

Noble plication

This procedure is time consuming and it involves release of all the involved coils of intestine are freed and adjacent coils are sutured with serosal sutures to form gentle curves of average length of 15-20 cm. The

mesentery of the corresponding bowel is sutured to prevent internal herniation.

Charles Phillips Operation

In this procedure, after adhesinolysis the bowel is placed in an orderly fashion and three synthetic sutures are passed through the mesentery of the plicated bowel, each doubled back upon itself and tied loosely. Stitches should pass a few centimetres away from the bowel wall. The resulting bowel should look like a pocket of sausages.

Intraluminal tube insertion

Several tubes are introduced into the small bowel through jejunostomy proximal to obstruction. Baker's tube or Miller Abbot tube is commonly used. The tubes are left in situ for 10-15 days. The tube is introduced to small bowel through proximal jejunostomy upto the caecum, where the balloon attached to this tube is distended and the proximal end of the tube is connected to a sterile drainage bag and drainage continued till the patient passes flatus. After 2 weeks the tube is gradually withdrawn after deflating the balloon under sedation. This jejunostomy site heals by granulation tissue. The tube facilitates the

formation of gentle smooth curved of small bowel. This technique was successful in relieving the obstruction in 80% of cases.

Internal hernias

When a segment of the small intestine herniates into one of the retroperitoneal fossae it is termed as internal hernias. This can occur in following sites:

- Supravesical hernia
- Foramen of Winslow
- Diaphragmatic hernia: Acquired/Congenital.
- Caecal or appendicaecal: Retroperitoneal fossae superior or retrocaecal
- A hole in the mesentery or mesocolon and defects in the broad ligament.
- Paraduodenal fossae: Right/left paraduodenal fossae.

INTUSSUSCEPTION

Figure 7: Intussusception

When one portion of the gut invaginates into the immediately adjacent loop, the condition is called as intussusception. Most of the time, it is proximal segment of bowel invaginates into the distal segment. It is one of the commonest cause in paediatric age group. It can also occur in adults. In adults, our 2/3rd rule may be applied. Two-thirds of adult intussusceptions are from known causes. Of these two-thirds are due to neoplasms. Of these neoplasms, two-thirds will be malignant.[22]

An intussusception constitutes following parts:

- Intussusception
- Intussuscepian
- Apex
- Neck

The types of intussusception are as follows:

Ileocolic (77%)

Ileo-ileocolic (12%)

Ileo-ileal (5%)

Colo-colic (2%)

Multiple (1%)

Retrograde (0.2%)

Others (2.8%)

In children intussusception can occur in any age but more commonly in the period between 3 months to 9 months when the weaning food is started. The incidence is higher in first-born child. It is more common in summer season.

Etiology

Most of the cases of intussusception occurs in paediatric age group. Majority of cases classified as idiopathic may be associated with acute gastroenteritis or URI or when the baby gets weaning started, it is believed to be due to hyperplasia of Payer's patches in the terminal ileum, polyps, Meckel's diverticulum, duplication. H.S. Purpura or appendix is the common lead points. Most patients with adult intussusception are benign and enteric origin and most sensitive diagnostic modality is abdominal CT scan.[25] Large bowel intussusception is more common in adults. Half of the cases are secondary to neoplasia either benign or malignant. Among benign conditions, fibroma, leiomyoma, polyps and submucous lipoma, Peutz Jeghers syndrome are commonest one.

Etiologically, adult intussusception could be categorized into four groups:

(1) tumor-related; (2) postoperative; (3) miscellaneous--Meckel diverticulum; and (4) idiopathic . The tumor-related intussusceptions were caused by benign tumors in five and malignant tumors in eight patients. Postoperative intussusceptions were related to various factors including suture lines, ostomy closure sites, adhesions, long intestinal tubes, bypassed intestinal segments, submucosal edema, abnormal bowel motility, electrolyte imbalance, and chronic dilatation of the bowel. The sites of involvement of intussusception were jejunogastric, jejunojejunal, ileoileal, ileocolic, and colocolic.

Clinical features: The typical presentation with intermittent pain abdomen. Usually it is sudden onset. During the attacks of pain the child cries with pallor over the face and becomes quite after few minutes. Vomiting and blood stained stools may be present. On examination during pain free period a sausage shaped lump may be palpable with concavity towards umbilicus, which becomes hard on palpation. Right iliac fossa on palpation is peculiarly empty (sign-de-dance). Patinet may pass a few normal motions before ‘red currant jully’ stool is passed.

Radiography

Plain X-ray may show multiple air fluid levels. Barium enema in ileo-colic type shows typical ‘pincer shaped’ or ‘coiled spring’ deformity or ‘pinch fork’ sign.

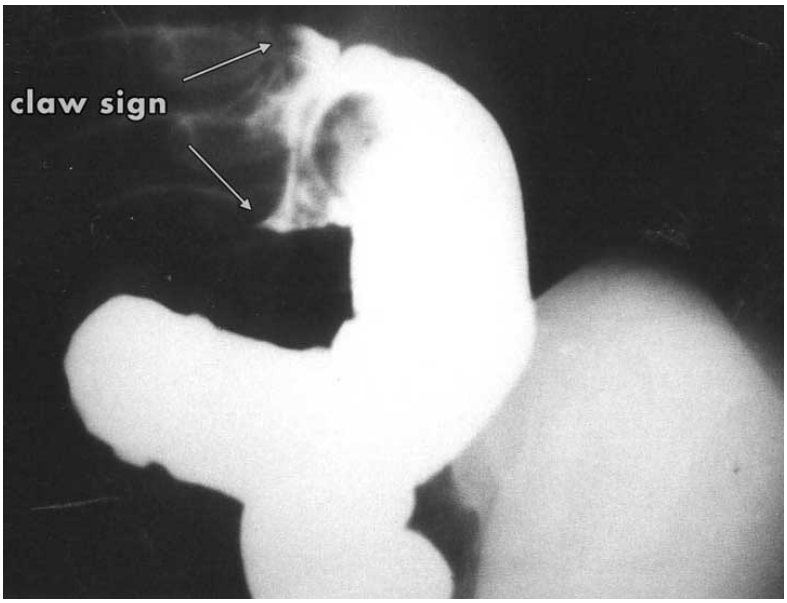


Figure 8: Plain X-ray showing claw sign

Treatment

Intravenous fluid administration should be started, decompression of small intestine through nasogastric suction is done. The reduction of intussusception can be done by the following ways, non-operative reductions: (1) Hydrostatic (2) Pneumatic.

1. Hydrostatic reduction: This type of reduction is a successful treatment in 60-70% of patients. This can be done in cases of less than 24 hours duration and good general condition, without any complications. Contraindications for hydrostatic reduction, signs of peritonitis, complete obstruction, perforation and duration >48 hours after onset of obstruction or signs and symptoms of gangrenous bowel.

Procedure

In this procedure, a rectal tube inserted and saline is allowed to run into the bowel for a while and tube is removed, fluid allowed to escape outside into a container. In initial attempts, blood stained fluid will come out, if the reduction has occurred, flatus and fecal matter is passed. If there is any doubt regarding the reduction, laparotomy is the next line of management.

2. Pneumatic reduction: The procedure is done under fluoroscopy. Gou et al. (1986) reported reduction rate of 90% using air pressure enemata. The maximum air pressure enema for infants is 80 mmHg. Gou et al. reported 6386 cases treated in China over 13 years with success rate of 95%.

Operative management

Laparotomy is done by taking right lower paramedian incision. Intussusception mass is identified and manual reduction is done. The reduction is effected by squeezing the apex retrogradely, at no time the segments should be pulled. Open method of inserting little fingers into the neck and separating the adhesions between the intussusception in difficult cases is tried (Copes method).

Indications for resection

When there is a polyp, tumour or Meckel's diverticulum or if the intussusception cannot be reduced by manipulative procedures or a frankly gangrenous bowel is there, bowel should be resected without attempting for reduction. The whole intussusception is resected with primary ileocolostomy. The ileum is cut proximal to the intussusception and the proximal end is anastamosed end to side with the transverse colon distal to the intussusception.

VOLVULUS

Volvulus is axial rotation of a portion of alimentary tract on its mesentery. It may be primary or secondary. Primary volvulus occurs due

to malrotation of the gut, abnormal mesenteric attachments or congenital bands. E.g. Caecal volvulus, volvulus neonatorum and sigmoid volvulus.

A secondary volvulus is due to actual rotation of a segment of bowel around an acquired adhesion or stoma.

When it is completed, it forms a close loop of obstruction with ischaemia of the segment involved.

PRIMARY VOLVULUS

Volvulus neonatorum

Predisposed by arrested rotation of the gut causing narrow mesentery of the small bowel and caecum. Symptoms and signs are pain abdomen, bilious vomiting. On laparotomy, the whole of the midgut has to be delivered out. Volvulus usually occurs in clockwise direction, which has to be untwisted and if any secondary obstructing lesions such as congenital bands (transduodenal band of ladd) have to be released and Ladd procedure is performed. In this procedure, duodenum and upper jejunum is repositioned on the right side of the abdomen.

Small bowel volvulus

It usually occurs in the lower ileum, it may be spontaneous after consumption of plenty of vegetable food material or it may be due to

adhesion passing to pelvic parities or pelvic organs in female. Treatment consists of laparotomy, derotation and causative adhesions has to be removed.

Caecal volvulus

It may occur when the right half of colon is lax and mobile. More common in female with classical features of obstruction like pain abdomen, nausea, vomiting, constipation. At first, it may be partial rotation and spontaneously released by passage of flatus and faeces. Barium enema may show a “bird beak” appearance and absence of barium in the caecum. At laparotomy volvulus can be treated by caecal needle decompression followed by fixation of caecum to right iliac fossa or if caecum is gangrenous or perforated volvulus is a serious condition in which a redundant sigmoid loop rotates around its narrow, elongated mesentry, producing ischaemia and necrosis of the sigmoid Sigmoid Colon, followed by rapid distension of the closed loop.[24] Volvulus of the pelvic colon is more common in Eastern Europe, Russia and Mrica. It is rare in USA and Western Europe. In India, it is common in northern states like Punjab, UP, MP, Bihar and Maharashtra. This is rotation of sigmoid loop of the colon over mesentery, commonly occurs in adults and elderly males and one of the commonest causes of large bowel obstruction.

Aetiology includes high residue diet with physical inactivity, chronic constipation, psychiatric problems right hemicolectomy is carried out.

Sigmoid volvulus[17], prolonged use of sedatives, addiction to laxatives acute psychotic drugs.

Males are more affected who are middle aged or elderly. More prevalent in under developed and malnourished people. Pathology may be due to long pelvic meso colon, adhesions over the sigmoid colon, overloaded pelvic colon.

The twist has odd turns, ½, 1½, 2½, etc. After the loop has rotated 1½ turns the veins involved in the torsion are compressed and the loop becomes greatly congested and if rotation is greater than 1½ times loop becomes gangrenous. It presents with signs and symptoms of acute large bowel obstruction, which may be intermittent initially and followed by passage of large quantity of faeces and flatus. Plain radiography of the abdomen shows an inverted “U” shaped shadow of distended loop (or coffee bean appearance) that runs from right to left with two fluid levels one in each loop.

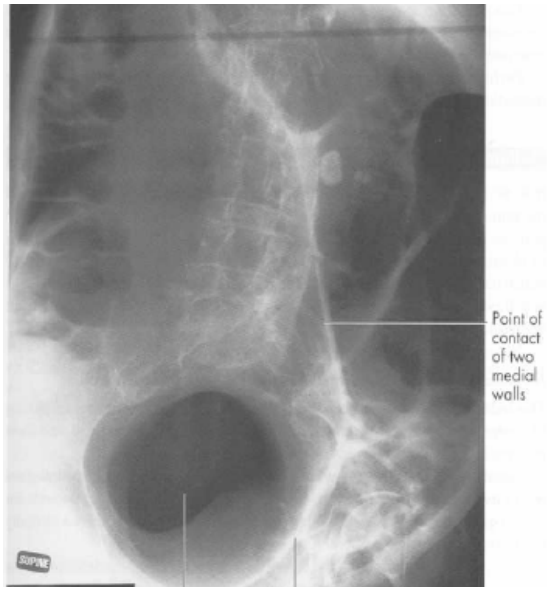


Figure 9: Radiological view of sigmoid volvulus

Management

Consists of conservative or surgical measures. Surgery is mandatory, if gangrene is suspected, resection anastamosis has to be carried out.

Conservative treatment

Sigmoidoscopy should be carried out, if the obstruction is reached a soft nectal tube is attempted to put into the twisted gut. This will immediately deflate the gut and surgery can be delayed until the patient is fit for surgery.

a. Oral medication

Oral liquid paraffin, 30-60 ml repeatedly given, it is said that liquid paraffin leads to relief of tension or oedema at the site of twist, thus loosening the constriction with further deflation, finally peristaltic wave may untwist the colon.

b. Enema

Untwisting phenomena may occur if enema given in early stages.

Surgical treatment

In patients with sigmoid volvulus who do not have peritonitis on presentation, recurrence preventing surgery may be performed with minimal mortality rates.[26]

After laparotomy through midline or left paramedian incision, the loop may be untwisted and deflation of the distended loop can be done through a stab incision under cover of purse string suture. The gangrenous bowel has to be resected and following procedure can be done.

a. Primary anastomosis after resection of gangrenous segment with proximal covering colostomy.

b. A Paul Mickulicz type double barrel colostomy.

c. Hartman's procedure: Proximal end colostomy with closure of the end of the distal colon or rectum in two layers and dropped into abdominal cavity. Later anastomosis can be done after one month.

Resective procedure

a. Resection with Paul Mickulicz procedures or Hartman's procedures followed by delayed anastomosis.

b. Resection and primary anastomosis: The redundant loop of sigmoid colon can be resected and primary anastomosis can be done.

The mortality of patients with sigmoid volvulus treated surgically is closely related to the disease stage, a prompt surgical timing, the patient functional status and his collaboration with clinicians is essential in order to define a correct diagnosis and treatment.[26]

Non-resective procedures

a. **Mesocolopexy:** Plication of sigmoid colon by various methods, which make the long mesocolon into more broad based and shorter length, which avoids the torsion of the sigmoid colon on its

mesentery Hall and Cragg's type, Prasad and Tiwari type had been advocated.

b. **Sigmoidopexy:** Involves fixation of sigmoid loop to the posterior wall of the abdomen or to the parietal peritoneum or to the transverse colon. The principle behind fixing the sigmoid loop to a various places is to curtail its free movements, which will avoid it from twisting.

c. **Extraperitonealization of sigmoid colon:** Sigmoid loop is placed in the space between lower abdominal peritoneum and abdominal musculature which may prevent or avoids rotation movements of the alimentary tract.

Ileosigmoid volvulus (compound volvulus)

Also known as ileosigmoid knotting. A rare condition where the long pelvic mesocolon allow the loop of ileum to twist over the root of mesocolon of sigmoid resulting in gangrene of either or both segments of the bowel. On laparotomy depending upon the viability of the bowel loop, decompressio n and if gangrenous, resection anastamosis of the sigmoid volvulus has to be done.

Treatment

Fluid Resuscitation and Antibiotics

Patients with intestinal obstruction are usually dehydrated and depleted of sodium, chloride, and potassium, requiring aggressive intravenous (IV) replacement with an isotonic saline solution such as lactated Ringer's. Urine output should be monitored by the placement of a Foley catheter. After the patient has formed adequate urine, potassium chloride should be added to the infusion if needed. Serial electrolyte measurements, as well as hematocrit and white blood cell count, are performed to assess the adequacy of fluid repletion. Broad-spectrum antibiotics are given prophylactically based on the reported findings of bacterial translocation occurring even in simple mechanical obstructions. In addition, antibiotics are administered as a prophylaxis for possible resection or inadvertent enterotomy at surgery.

Tube Decompression

Apart from IV fluid resuscitation, another important adjunct to the supportive care of patients with intestinal obstruction is nasogastric suction. Nasogastric suction with a Levin tube empties the stomach, reducing the hazard of pulmonary aspiration of vomitus and minimizing

further intestinal distention from preoperatively swallowed air. The use of long intestinal tubes (e.g., Cantor or Baker tubes) can be used. Patients with a partial intestinal obstruction may be treated conservatively with resuscitation and tube decompression alone. Enteroclysis can assist in determining the degree of obstruction, with higher-grade partial obstructions requiring earlier operative intervention. Although an initial trial of nonoperative management of most patients with partial small bowel obstruction is warranted, it should be emphasized that clinical deterioration of patient or increasing small bowel distention on abdominal radiographs during tube decompression warrants prompt operative intervention.

Operative Management

The patient with a complete small bowel obstruction requires operative intervention. A nonoperative approach to selected patients with complete small intestinal obstruction has been proposed by some, who argue that prolonged intubation is safe in these patients provided that no fever, tachycardia, tenderness, or leukocytosis is noted. Nevertheless, one must realize that nonoperative management of these patients is undertaken at a calculated risk of overlooking an underlying strangulation obstruction and delaying the treatment of intestinal strangulation until

after the injury becomes irreversible. The Patients with intestinal obstruction secondary to an adhesive band may be treated with lysis of adhesions. Great care should be used in the gentle handling of the bowel to reduce serosal trauma and avoid unnecessary dissection and inadvertent enterotomies. The hernia may be a spontaneous groin, epigastric or paraumbilical hernia or it may be an incisional or parastomal hernia, the surgical approach and initial dissection is similar to that in elective hernia repair.[30]Incarcerated hernias can be managed by manual reduction of the herniated segment of bowel and closure of the defect. The treatment of patients with an obstruction and a history of malignant tumors can be managed by making a simple bypass of the obstructing lesion, by whatever means, may offer the best option rather than a long and complicated operation that may entail bowel resection. An obstruction secondary to Crohn's disease will often resolve with conservative management if the obstruction is acute. If a chronic fibrotic stricture is the cause of the obstruction, then a bowel resection or strictureplasty may be required. At the time of exploration, it can sometimes be difficult to evaluate bowel viability after the release of a strangulation. If intestinal viability is questionable, the bowel segment should be completely released and placed in a warm, saline-moistened sponge for 15 to 20 minutes and then reexamined. If normal color has

returned and peristalsis is evident, it is safe to retain the bowel. In difficult borderline cases, fluorescein fluorescence may supplement clinical judgment. Another approach to the assessment of bowel viability is the so-called second look laparotomy within 18 to 24 hours.

TUBERCULOSIS OF INTESTINE

Intestinal obstruction is the most common complication in the small bowel, affecting 60% of the patients with tuberculous enteritis. Common site is ileum, proximal colon and peritoneum. Approximately, 75% of patients with tuberculosis enteritis have involvement of the distal small bowel and ileocaecal region.[31]

Intestinal obstruction is the most common complication in the small bowel, affecting 60% of the patients with tuberculum enteritis.

There are two principal types:

1. Hyperplastic tuberculosis

The infection establishes itself in lymphoid follicles and the resulting chronic inflammation causes thickening of the intestinal wall and narrowing of the lumen. Pain abdomen with intermittent diarrhoea are usual symptoms. Sometimes mass in the right iliac fossa. In non-

obstructed patients, treatment is with antituberculosis drugs and in patients with intestinal obstruction the presentation will be subacute intestinal obstruction which should be managed by resection of ileocaecal segment. When the patient presents with acute intestinal obstruction is treated with ileotransverse anastomosis.

2. Ulcerative tuberculosis

In this condition there are multiple ulcers in the terminal ileum, serosal thickening, reddened and covered in tubercle. These patients are treated with antitubercular treatment, presents with intestinal obstruction secondary to stricture. There are treated by stricturoplasty or by resection of segment of bowel which contains multiple stricture or long segment stricture.

Other forms of strictures

Crohn’s disease is among the most common aetiology of small intestinal stricture. Certain drugs are known to cause mucosal ulceration and strictures, drugs most notably are enteric coated potassium chloride preparations and NSAIDs. Radiation therapy for intraperitoneal malignancy can lead to strictures, mesenteric ischaemia can lead to stricture formation, the distal ileum being at greatest risk as the ileocolic

artery is the last branch of superior mesenteric artery. Various neoplasms including carcinoma, carcinoid, lymphoma can cause stricture within small intestine. Resection is the treatment whenever technically feasible.

LARGE BOWEL OBSTRUCTION[27]

Colorectal cancer is the single most common cause of large intestinal obstruction in the United States, whereas colonic volvulus is the more common cause in Russia, Eastern Europe, and Africa. Intraluminal causes of colorectal obstruction include fecal impaction, inspissated barium, and foreign bodies. Intramural causes, include inflammation (diverticulitis, Crohn's disease, lymphogranuloma venereum, tuberculosis, and schistosomiasis), Hirschsprung's disease (aganglionosis), ischemia, radiation, intussusception, and anastomotic stricture. Extraluminal causes include adhesions (the most common cause of small bowel obstruction, but rarely a cause of colonic obstruction), hernias, tumors in adjacent organs, abscesses, and volvulus.

Etiology

Carcinoma of the colon

Carcinoma is the most frequent cause of large-bowel obstruction in developed countries . The left colon is the most likely site of obstruction

and the extraperitoneal rectum the least. Signs of partial obstruction progress to those of complete obstruction when the narrowed colonic lumen is occluded by a fecal bolus. Since the right colon has semiliquid contents and a relatively wide lumen, obstruction occurs late in this segment and may be acute in its presentation, especially if the ileocecal valve is competent. The operative risk is increased considerably when perforation is present.

SIGNS AND SYMPTOMS

The signs and symptoms of large bowel obstruction depend on the cause and location of the obstruction. Cancers arising in the rectum or left colon are more likely to obstruct than those arising in the more capacious proximal colon. Regardless of the cause of the blockage, the clinical manifestations of large bowel obstruction include the failure to pass stool and flatus associated with increasing abdominal distention and cramping abdominal pain. Colonic obstruction is associated with potentially serious complications such as perforation, only 4% of the tumours of the colon presnt with the perforation of the bowel, and the timing and selection of appropriate operative procedures are important. Symptoms can develop slowly and progressively or fulminantly. Among adults, elderly people are usually affected. The sigmoid colon is the usual site: this portion of

the intestine is thick walled, not particularly distensible, and comparatively narrow.

MANAGEMENT

All patients with complete acute large bowel obstruction require prompt surgical intervention and should not undergo a trial of nonoperative management. Nasogastric decompression is also important in patients with a large bowel obstruction to decrease the amount of air and gastric contents delivered to the bowel. Nasogastric decompression will help relieve intraluminal pressure, prevent further dilation of the proximal bowel, and possibly decrease the risk of perforation.Antibiotics targeted at both skin and colonic flora should be administered.

Exploration in patients with large bowel obstructions is best performed through a low midline incision. Patients with large bowel obstructions should be placed in the lithotomy or modified lithotomy position if access to the anus is anticipated. Obstructing lesions of the cecum and ascending colon should be resected via right hemicolectomy, usually with a primary anastomosis. Lesions in the transverse colon should be managed with an extended right hemicolectomy and again, with a primary anastomosis. Proximal diversion with an end ileostomy is not necessary in all patients; however, proximal diversion should be

considered when there is any concern about bowel viability, if the patient is unstable, or in the case of substantial peritoneal contamination or peritonitis.

The management of obstructing lesions in the descending and sigmoid colon is a more classic approach with a Hartmann's procedure of segmental resection of the affected colon, an end colostomy, and a blind distal pouch or mucous fistula. An end colostomy at the time of operation is safe and may decrease the incidence of perioperative complications compared to an on-the-table bowel preparation with primary anastomosis.

Another option to consider in the early management of the patient with an obstructing lesion in the large bowel is the use of a self-expanding intraluminal metal stent (SEMS) to allow immediate colonic decompression and the ability to perform elective mechanical bowel preparation.The use of SEMS is becoming widely available and it can be a useful tool for the surgeon managing a large bowel obstruction. In experienced hands, a SEMS can be placed successfully in about 90% of patients with low complication rates. A SEMS can avoid the need for urgent or emergent operation by intraluminally decompressing the distended proximal colon and allowing distal passage of stool. A SEMS is also useful when palliating patients who might not tolerate surgical

diversion or those with unresectable disease and a limited survival. With a locally advanced obstructing rectal cancer, after placement of a SEMS, the patient can undergo neoadjuvant therapy followed by surgical resection, again increasing the chances for a successful one-stage operation.

The cause of the obstruction needs to be managed individually. Thus a hernia is repaired once the obstructed colon is reduced, whereas an intussusception is reduced, with resection of the involved colon if necessary. Because it is so common, fecal impaction should be ruled out in institutionalized or debilitated elderly patients. The cecum must be visualized to assess its viability. If an anastomosis is to be performed, the criteria for a good outcome must be met.

ADYNAMIC OBSTRUCTION

Adynamic obstruction, in which peristalsis may be absent as in paralytic ileus or it may be present in a non propulsive form as in mesenteric vascular occlusion or pseudoobstruction.

Paralytic Ileus

A paralytic ileus is defined as functionally impaired transit of intestinal contents because of decreased peristaltic activity of the

gastrointestinal tract, in the absence of mechanical obstruction.34 An ileus can result from a number of causes, including drug induced, metabolic, neurogenic, and infectious.

Causes of Ileus

- Idiopathic
- After laparotomy
- Metabolic and electrolyte derangements (e.g., hypokalemia, hyponatremia, hypomagnesemia, uremia, diabetic coma)
- Drugs (e.g., opiates, psychotropic agents, anticholinergic agents)
- Intra-abdominal inflammation
- Retroperitoneal hemorrhage or inflammation
- Intestinal ischemia
- Burns
- Strokes

Pharmacologic agents that can produce an ileus include anticholinergic drugs, autonomic blockers, antihistamines, and various psychotropic agents, such as haloperidol and tricyclic antidepressants. One of the more common causes of drug induced ileus in the operative patient is the use of opiates, such as morphine or meperidine. Metabolic

causes of ileus are common and include hypokalemia, hyponatremia, and hypomagnesemia. Other metabolic causes include uremia, diabetic coma, and hypoparathyroidism. Neurogenic causes of an ileus include postoperative ileus, which occurs after abdominal operations. Spinal injury, retroperitoneal irritation, and orthopedic procedures on the spine or pelvis can result in an ileus. Finally, a number of infectious causes can result in an ileus; common infectious causes include pneumonia, peritonitis, and generalized sepsis from a nonabdominal source.

Patients often present in a manner similar to those with a mechanical small bowel obstruction. Abdominal distention, usually without the colicky abdominal pain, is the typical and most notable finding. Nausea and vomiting may occur but may also be absent. Patients with an ileus may continue to pass flatus and diarrhea, and this may help distinguish these patients from those with a mechanical small bowel obstruction.

Radiologic studies may help to distinguish ileus from small bowel obstruction. Plain abdominal radiographs may reveal distended small bowel as well as large bowel loops.

The treatment of an ileus is entirely supportive with nasogastric decompression and IV fluids. The most effective treatment to correct the

underlying condition may be aggressive treatment of the sepsis, correction of any metabolic or electrolyte abnormalities, and discontinuation of medications that may produce an ileus. Drugs that block sympathetic input (e.g., guanethidine) or stimulate parasympathetic activity (e.g., bethanechol or neostigmine) can be used. In addition, hormonal manipulation, using cholecystokinin or motilin, IV erythromycin have been but ineffective. Alvimopan, a peripherally acting muopioid-receptor antagonist, appears to decrease the time to recovery of bowel function after major abdominal operationsc ileus

Acute Colonic Pseudo-Obstruction[39]

Acute colonic pseudo-obstruction also known as Ogilvie's syndrome is an often painless paralytic ileus of the large bowel characterised by rapidly progressive abdominal distension. Acute pseudo-obstruction of the colon is often suspected based both on abdominal radiography and the clinical setting, but it remains a diagnosis of exclusion. When peritonitis or perforation is present or ischemia is suspected, the patient requires semi-emergent operative intervention, but only after appropriate resuscitation. Exclusion of a mechanical obstruction can be accomplished via either a careful, complete colonoscopy minimizing air insufflation, or by demonstrating free

retrograde flow of contrast without obstruction to the cecum on water-soluble enema. These water-soluble contrast enemas can also be therapeutic; some patients achieve decompression after contrast instillation because of stimulation of defecation. Initial management of pseudo-obstruction is the same as that for small bowel obstruction and includes placement of a nasogastric tube, nothing by mouth, rehydration, correction of electrolyte abnormalities, and if possible, discontinuation of narcotics. The decision to pursue colonic decompression is based in part on the cecal diameter as determined by abdominal radiographs, on symptomatology, and on the duration of obstruction. In general, the maximum accepted safe cecal diameter is 12 cm; above this diameter, the risk of perforation increases substantially. When the cecum is less than 12 cm in diameter and the patient is not distressed, initial treatment should be continued, as immediate decompression is not necessary. If at any time during therapy the patient develops peritonitis or signs of ischemia, emergent surgical therapy is indicated.

Acute colonic pseudo-obstruction, with a markedly dilated proximal colon; endoscopy confirmed no distal obstruction, When the cecal diameter exceeds 12 cm or the patient has a significant amount of abdominal discomfort, decompression is indicated. The two methods of colonic decompression include intravenous neostigmine and colonoscopic

decompression.[40,41] Neostigmine is the first line of treatment in the patient without contraindications. Neostigmine has been shown to be effective for treatment of acute colonic pseudo-obstruction, achieving success in about 90% of patients.

Colonoscopic decompression, once the first line of therapy for acute colonic pseudo-obstruction, is now considered the second-line treatment for patients with uncomplicated pseudo-obstruction. Colonoscopic decompression is indicated in those patients with contraindications to neostigmine administration or those who have failed neostigmine therapy. Another role for colonoscopy is to exclude a source of mechanical obstruction, in which case it may be both diagnostic and therapeutic. Most surgeons, however, do not place a long colonic tube at the time of first colonoscopic decompression, but do leave a tube should the patient require another colonoscopic decompression for recurrent obstruction. When a colonic tube is deemed necessary, the tube is left within the dilated segment, not just in the rectum.

Operative intervention is indicated when signs and symptoms of perforation or ischemia are present or when conservative measures have failed. In the presence of ischemia or when splitting of the teniae coli is evident, resection of the involved segment, usually the cecum and

ascending colon, is warranted. Primary anastomosis can often be performed without a bowel preparation on the right side of the colon, as long as the residual tissues and medical condition of the patient are favorable.

When surgical intervention is necessary after failed medical therapy and no evidence of ischemia exists, some authors recommend a surgical cecostomy tube be placed. The tube placed should have a large diameter (>32F) and be flushed frequently to prevent obstruction with stool, which can be quite problematic.

MATERIAL AND METHODS

The materials for the clinical study of intestinal obstruction were collected from cases admitted to various surgical wards in RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL from June2012 to December 2012, fifty cases of intestinal obstruction have been studied. Patients belonged to the age groups ranging from 12 years to 85 years, paediatric age group is excluded from this study. The criteria for selection of cases was based on clinical history, physical findings, radiological and haematological investigations.

Patients who were having subacute intestinal obstruction treated conservatively were excluded from the study, and only those cases of acute intestinal obstruction which were managed surgically were studied to establish the pathology of intestinal obstruction with an aim to know the mode of presentation, physical findings, radiological and haematological findings, operative findings and outcome of acute intestinal obstruction.

After the admission of the patient, clinical data were recorded as per Proforma .The diagnosis mainly based on clinical examination and often Study divided into supported by haematological and radiological examinations.

Methods

a. Clinical study

b. Investigations

c. Treatment

Study was conducted under the following headings:

a. History taking

b. Physical examination

c. Laboratory examination

d. Radiological examination – Plain X-ray erect abdomen

e. Ultrasound examination in selected cases

f. Surgical treatment and results

g. Follow-up

a. History taking

A complete history was obtained from the patient and the complaints entered in the proforma in a chronological order. Each complaint in the history of presenting illness was documented in detailed enquiry.

b. physical examination

(i) General physical examination – evidence of dehydration and the severity of it were looked into it and vital parameters were recorded.

(ii) Local examination – Abdominal examination was done under standard headings inspection, palpation, percussion and palpation. Per rectal examination was done and findings were noted.

(iii) Systemic examination – All other systems were examined carefully to rule outassociated anomalies and to assess the fitness for surgery.

c. Laboratory examination

(i) Haemoglobin

(ii) TC & DC

(iii) BT and CT

(iv) Blood grouping and Rh typing

(v) Urine for albumin and microscopy

d. Radiological examination

Erect abdomen X-ray done in all cases, barium enema and ultrasound examination in selected cases.

SURGICAL MANAGEMENT

Immediately after the admission along with above procedure resuscitation with IV fluids especially ringer lactate and normal saline infusion started till the hydration and urine output become normal. Nasogastric decompression with Ryles tube carried out and antibiotic prophylaxis started. And close observation of all bedside parameters (like pulse rate, BP, RR, urine output, urine output, abdominal girth, bowel sounds and tenderness and guarding) was done. Blood transfusion was given in required cases. Patients who showed reduction in abdominal distension and improvement in general condition especially in individuals with postoperative adhesions conservative management was confined (by extending the supportive treatment) for next 24 hours, those who showed improvement by moving bowels, reduction in pain/tenderness were decided for conservative treatment, such individuals are excluded from this study.

Patients with clear-cut signs and symptoms of acute obstruction were managed appropriate surgical procedure after resuscitation. Surgery adopted and criteria for deciding the procedure were noted, e.g. release of a band or adhesion, reduction and caecopexy for intussusception, resection and anastamosis for gangrenous bowel and release and repair

for strangulated obstruction. Histopathological examination of the specimen of resection/biopsy was done whenever necessary.

The postoperative period was monitored carefully and all parameters were recorded hourly or four hourly basis depending upon the patients general condition and toxemia. Postoperatively Ryle's tube aspiration, intravenous fluids and antibiotics were administered. Any complications noted and treated accordingly.

Postoperative follow up after the discharge of patients was done in majority of the patients upto 6 months. Most of the patients did not come for follow up after one or two visits.

The results are tabulated stressing on following points age, sex, symptoms, examination findings, investigations, abnormalities, probable causative factors, operative findings and operative procedure adopted and complications if any.

Statistical Methods:

Statistical Data were analysed by using percentages and proportions .software namely Microsoft word and Excel have been used to generate graphs, tables, etc.

DATA ANALYSIS AND RESULTS

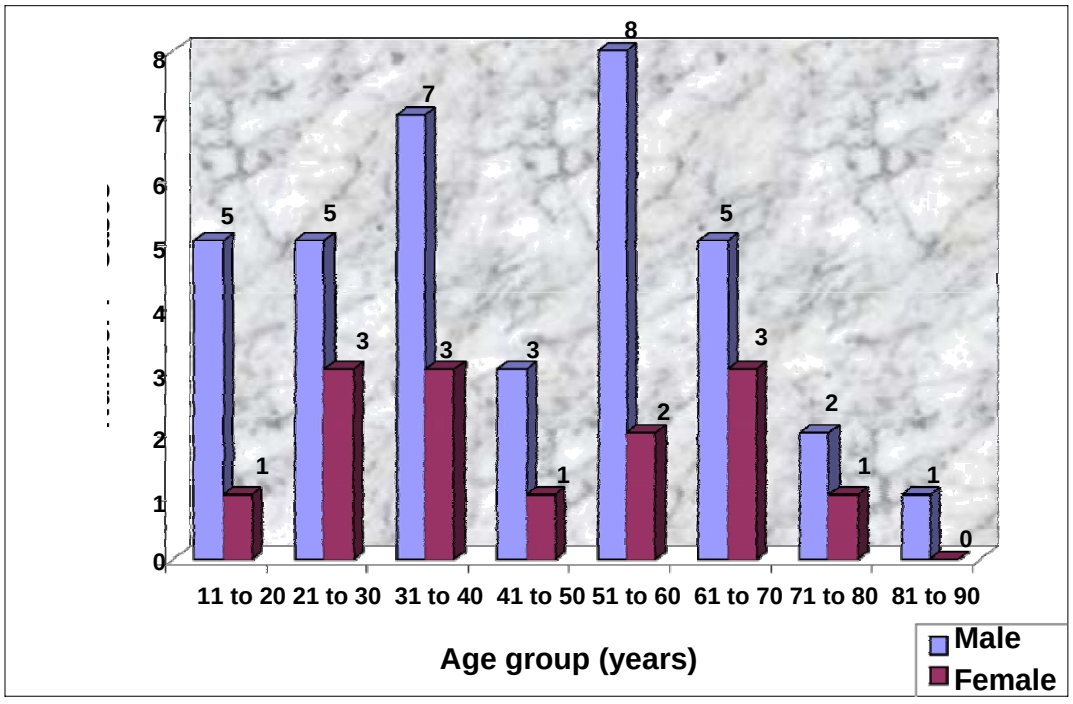
The incidence of acute intestinal obstruction in adult age group was studied from the cases admitted in Department of Surgery of RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL from June2012 to december 2012, fifty cases of intestinal obstruction have been studied. data regarding the symptoms and signs and laboratory investigations has been adopted in 50 cases during the study period.

During the period of 6 months, the total number of admissions in surgery were 12,233 cases. Of which 228 cases of acute intestinal obstruction were treated during these period which comprise 1.9% of total number of admissions among these surgically treated cases, 50 cases were randomly selected for the present study. Total number of emergency surgeries done in the department of Surgery were 1,569 and acute intestinal obstruction in this group consisted of about 14.53% of these surgeries.

Table 1: Age incidence

Age (years)	Male	Female	Total
11 to 20	5	1	6
21 to 30	5	3	8
31 to 40	7	3	10
41 to 50	3	1	4
51 to 60	8	2	10
61 to 70	5	3	8
71 to 80	2	1	3
81 to 90	1	0	1
Total	36	14	50

Figure 10: Age incidence

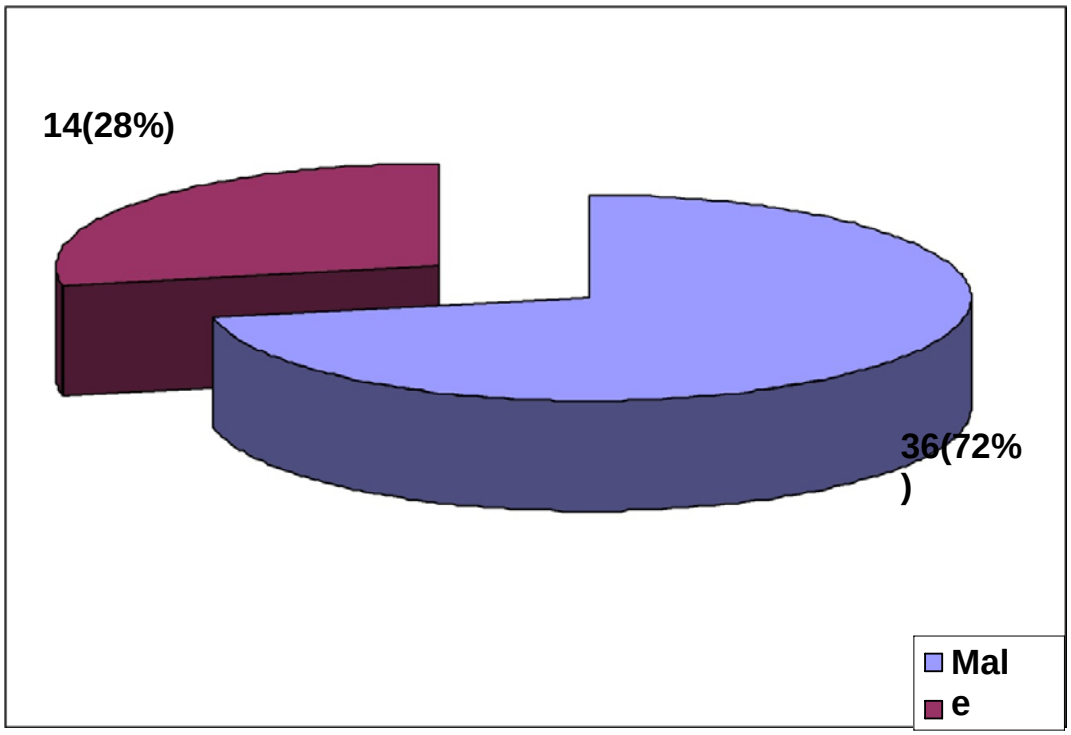


As per the above table and bar chart, the maximum incidence in the present study group is 31-40 and 51-60 with each 10 cases out of 50 cases

Table 2: Sex incidence

Sex	Number of cases	Percentage
Male	36	72
Female	14	28

Figure 11: Sex incidence



Male patients were more commonly affected when compared to females in the ratio of 4:1 in the above table.

Table 3: Socio-economic status

Socio-economic	Number of cases	Percentage
Poor	38	76
Middle	12	24
Upper	0	0
Total	50	100

Figure 12: Socio-economic status

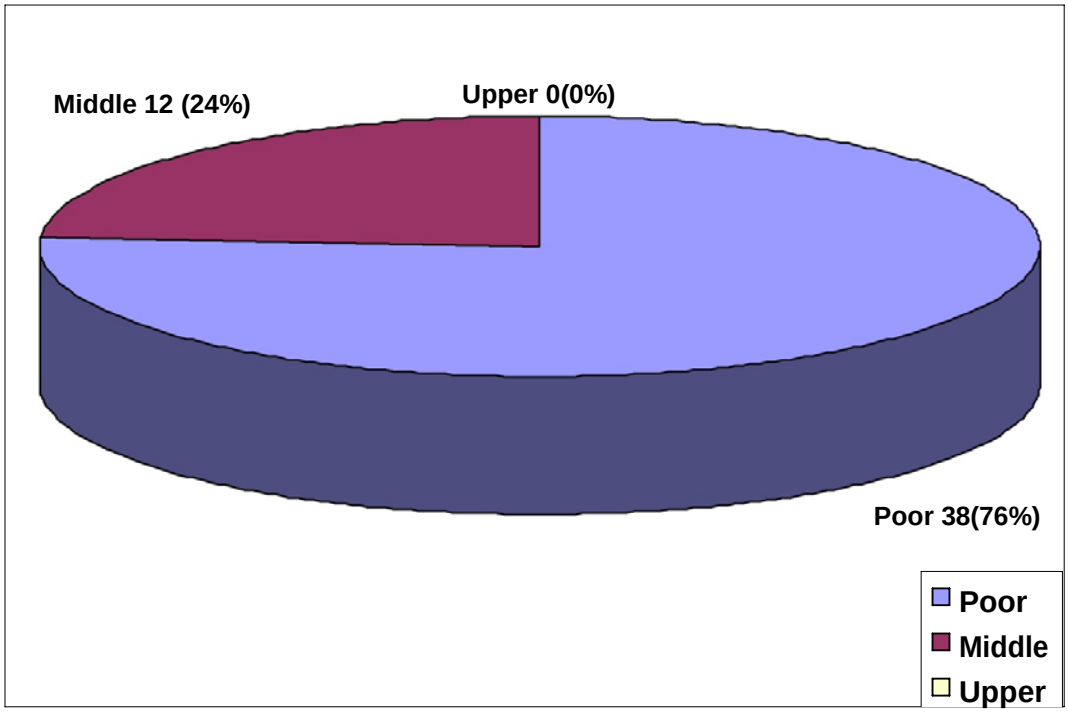
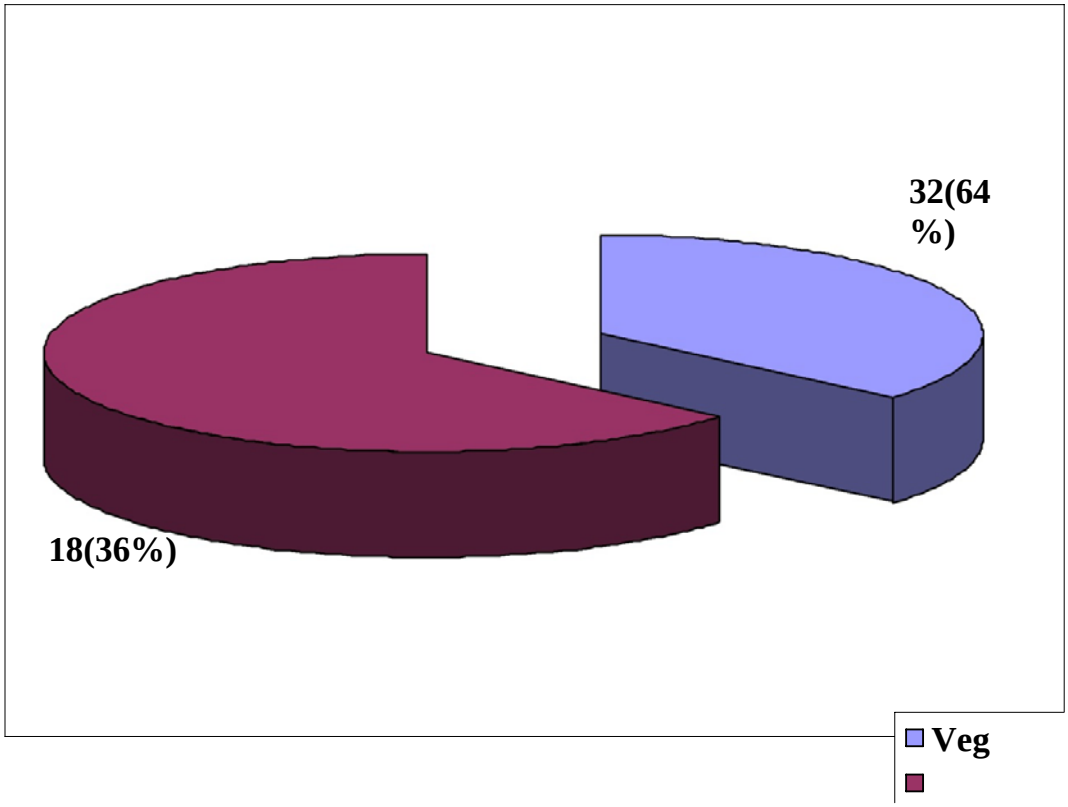


Table 4: Diet

Diet	Number of cases	Percentage
Veg	18	36
Non-veg	32	64
Total	50	100

Figure 13: Diet

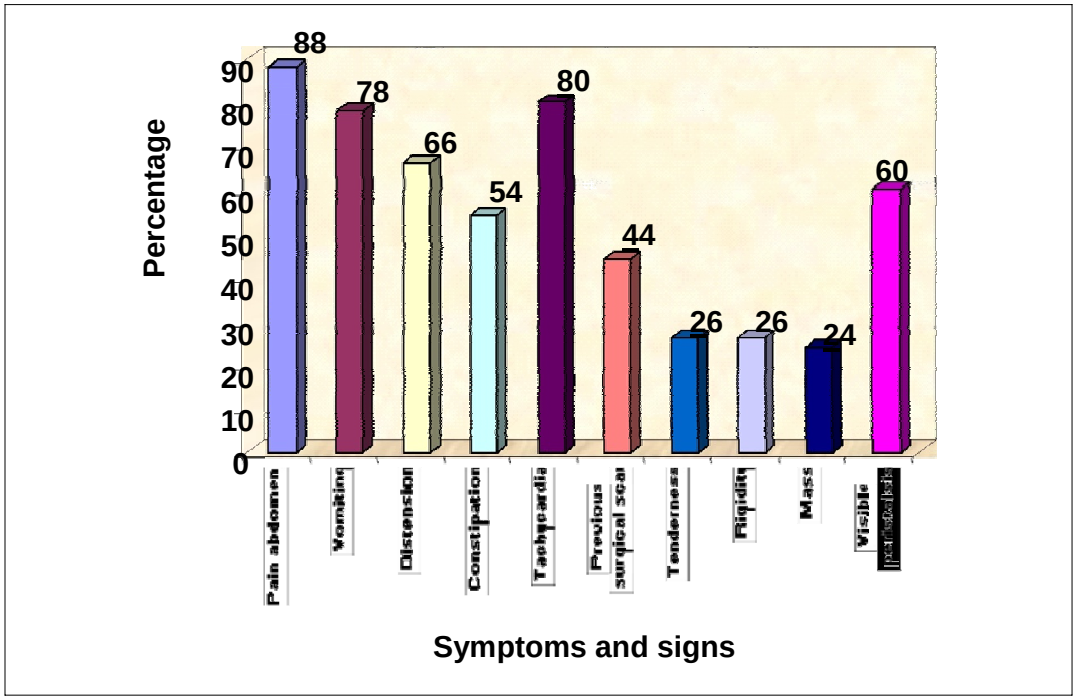


In the present study of 50 cases, 32 patients were taking non-vegetarian which contains more of fatty diets. The remaining 18 patients were vegetarian which oftenly contained high fibre diet.

Table 5: Symptoms and signs

Symptoms and signs	Number of cases	Percentage
Pain abdomen	44	88
Vomiting	39	78
Distension	33	66
Constipation	27	54
Tachycardia	40	80
Previous surgical scar	22	44
Tenderness	13	26
Rigidity	13	26
Mass	12	24
Visible peristalsis	30	60

Figure 14: Symptoms and signs



The present study the most common symptoms were pain abdomen (88%) and vomiting (78%), and the most common signs were tachycardia (80%) and visible intestinal peristalsis (60%).

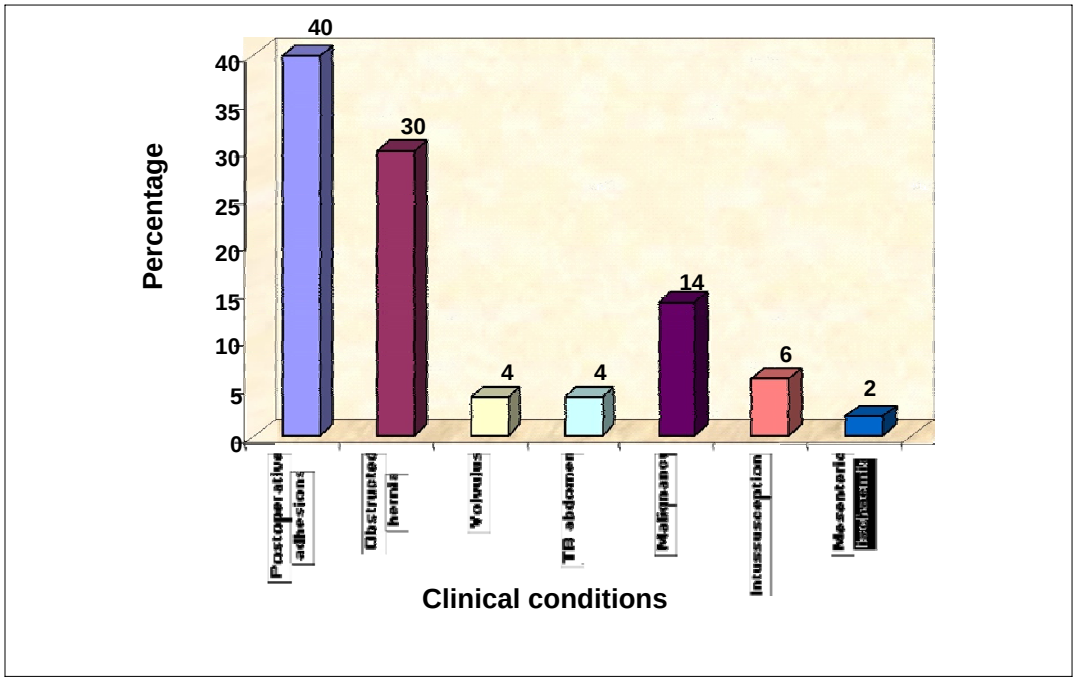
INCIDENCE OF DIFFERENT AETIOLOGY

The incidence of different etiologies of intestinal obstruction in the present series are as follows.

Table 6: Causes of intestinal obstruction in adults

Clinical condition	Number of cases	Percentage
Postoperative adhesions	20	40
Obstructed hernia	15	30
Volvulus	2	4
TB abdomen	2	4
Malignancy	7	14
Intussusception	3	6
Mesenteric ischaemia	1	2
Total	50	100

Figure 15: Causes of intestinal obstruction in adults

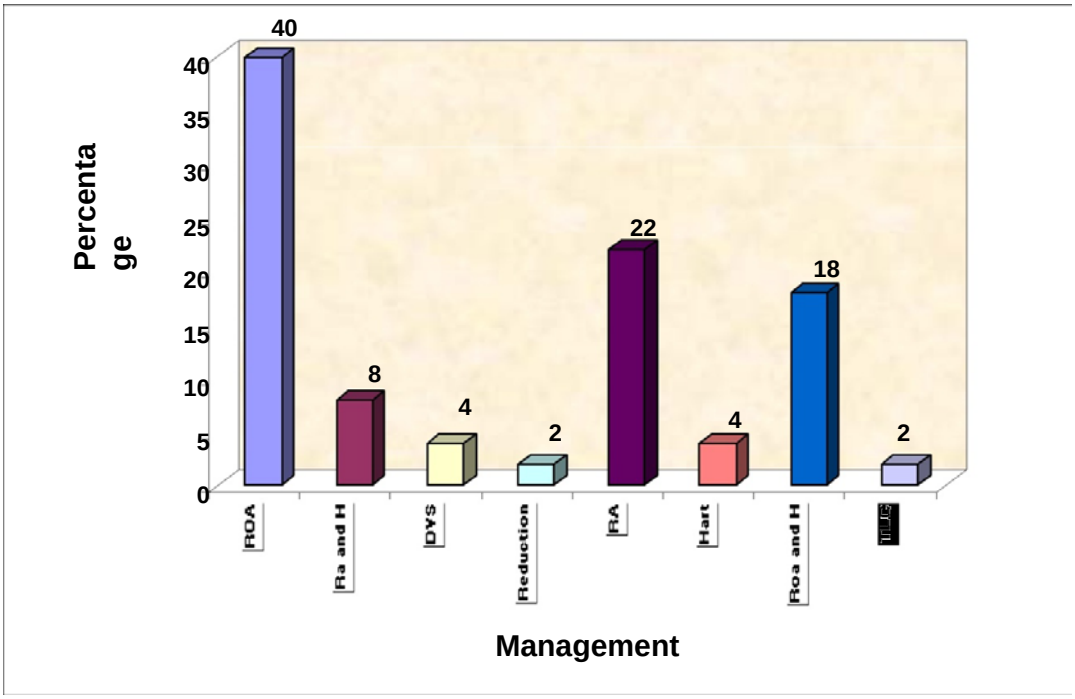


The most common cause of intestinal obstruction in our study was postoperative adhesions. The next common was obstructed hernia. Other conditions include volvulus, intussusception, tuberculosis, malignancy, mesenteric ischaemia, in descending frequency.

Table 7: Management

Management	Number of cases	Percentage
ROA	20	40
Ra and H	4	8
DVS	2	4
Reduction	1	2
RA	11	22
Hart	2	4
Roa and H	9	18
TLC	1	2
Total	50	100

Figure 16: Management

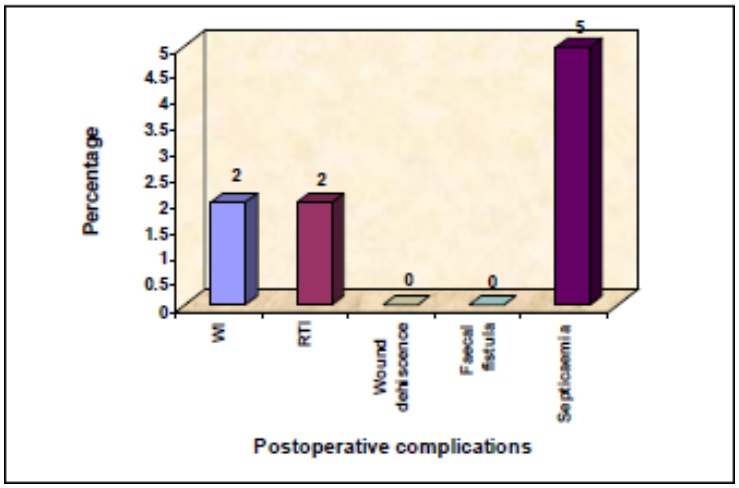


In our study of 50 cases as accordingly with the aetiology the management and the surgical procedure was done as shown in the table and pie diagram. Release of adhesions was done in 40% of cases, resection anastamosis in 22% of cases and release of adhesion with herniography done in 18% of the cases.

Table 8: Postoperative complications

Postoperative complications	Number of cases
WI	2
RTI	2
Wound dehiscence	-
Faecal fistula	-
Septicaemia	5

Figure 17: Postoperative complications



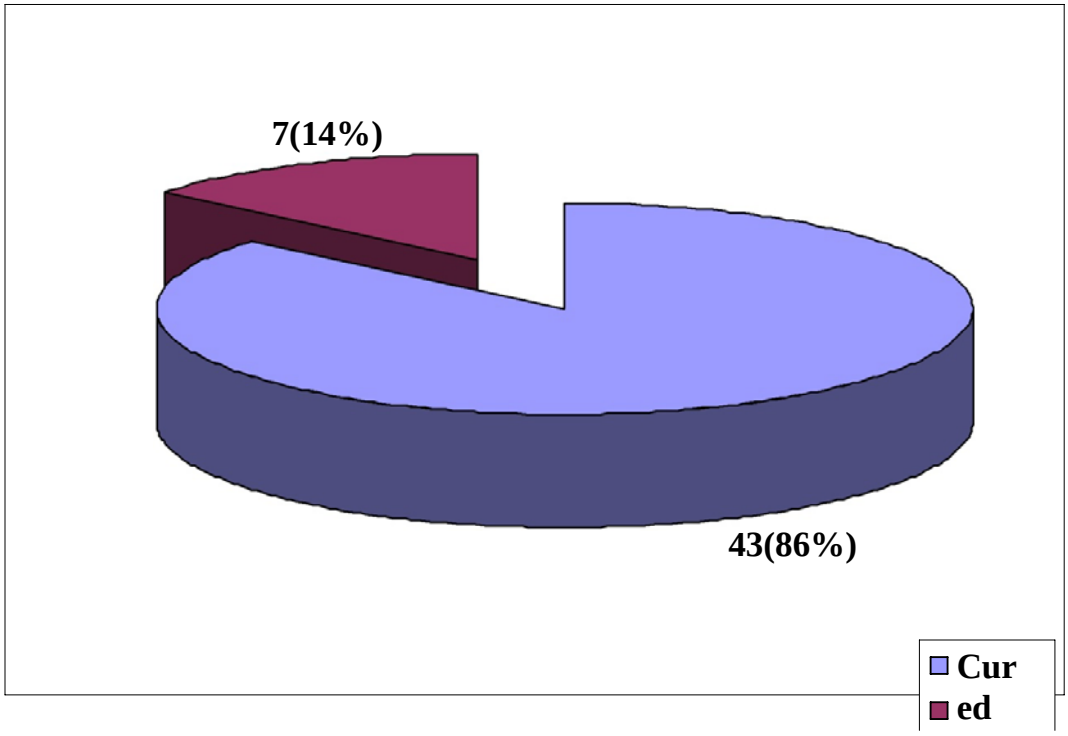
In the present study group there were 5 cases of septicemia, 2 cases of respiratory tract infection and 2 cases of wound infection.

Mortality

Table 9: Mortality

Mortality	Number of cases	Percentage
Cured	43	86
Dead	7	14

Figure18 : Mortality



Dead

In the present study of 50 cases, about 7 patients died with the percentage of 14%. The majority of deaths due to complications like septicemia, peritonitis, respiratory infection. In the present study 7 persons died during postoperative period. The analysis of cause of death is shown below.

Age and sex	Symptoms prior to admission	Operative findings	Operative procedure	Cause of Death
75/F (Case No. 8)	3 days	Carcinoma sigmoid colon	Resection and anastamosis	Septicaemic Shock
72/M (Case No.11)	8 days	Carcinoma Rectum	Hartman's procedure	RTI
65/M (Case No.21)	5 days	Mesenteric ischaemic	Resection anastamosis	Septicaemic Shock
45/M (Case No.36)	3 days	Carcinoma caecum	Resection and anastamosis	RTI
38/F (Case No. 37)	5 days	Carcinoma ovary with sigmoid colon infiltration	Transverse loop colostomy	Septicaemic Shock
63/M (Case No.39)	3 days	Carcinoma Rectum	Hartman's procedure	Septicaemia
55/M (Case No.43)	4 days	Carcinoma colon	Resection and anastamosis	Septicaemia

Table 10: Follow-up status

Follow-up complications	Follow-up status		
	One month	3rd month	6Th Month
A. Wound infection	1	Nil	Nil
B. Septicemia	Nil	Nil	Nil
C. Enterocutaneous Fistula	Nil	Nil	Nil
D. Prolonged ileus	Nil	Nil	Nil
E. Fever	2	3	Nil
F. Respiratory infection	2	1	Nil
G. Death	Nil	Nil	Nil

DISCUSSION

Acute intestinal obstruction continues to be the most common surgical emergency. In our study a total number of 12233 patients were admitted in the surgery department from January 2009 to June 2010. A total of 228 patients presented with features of acute intestinal obstruction. Among these 50 cases of operated cases were randomly selected for the present study.

Disease Incidence

In our clinical study incidence of acute intestinal obstruction is 1.9% of total surgical cases. In Souvik Adhikari et al. series incidence was 9.87% of total surgical cases. In Bhargava Anderson's series incidence was 3% of total surgical cases. The commonest cause was found to be postoperative adhesions followed by obstructed/ strangulated inguinal hernia, malignancy, intussusception, volvulus, tuberculosis and mesenteric ischaemia. Although in developing countries like India, the commonest cause used to be obstructed/strangulated hernia, in our study commonest cause was adhesions followed by obstructed/strangulated hernia as second cause. The decrease in the incidence of obstructed hernias indicate a changing trend towards early operation before hernia gets complicated. The data of the present series is comparable to Souvik

Adhikari series, Cole series and Jahangir-Sarwar Khan series. Souvik Adhikari et al. (2010) [42.]g reported an incidence of 9.87%, Bhargava and Anderson series reported an incidence of 3%. In our hospital 1569 cases of total emergency surgeries were done in June 2009 to June 2010, of which 228 cases of intestinal obstruction comprising of 14.3% incidence were present. Among these 50 cases were selected as random study group.

Age Incidence

Intestinal obstruction although occurs in all age groups, the age spectrum in our clinical study, with the spectrum age group of 15 years to 85 years. The study showed the peak incidence is in the age group 31-40 of 20% and 51-60 years of 20% which is comparable with the previous study groups Souvik Adhikari et al., Cole GJ et al. group, which are almost similar to our clinical study of intestinal obstruction. The mean age is our current study is 45 years where as Souvik Adhikari et al.[42] shows mean age of 44 years, Jahangir Sarwar Khan[43] series shows mean age is 33 years

These studies are almost comparable with our current study.

Table 11: Age incidence of intestinal obstruction in different studies

Age group	Cole GJ[44]	Souvik Adhikari[42]	Harban Singh[45]	Present study
12-20	10%	9%	10%	12%
21-30	10%	11%	16%	16%
31-40	18%	15%	18%	20%
41-50	16%	24%	15%	8%
51-60	15%	13%	10%	20%
61-70	16%	20%	20%	16%
71-80	9%	8%	5%	6%
81-90	6%	4%	4%	2%

Sex Incidence

In Souvik Adhikari et al.[42] study male to female radio was 4:1. In Osuigwe AN et al. study male to female ratio was 2:1. In the present study male to female ratio is 4:1.

Etiology

The cause of intestinal obstruction differs from different geographical locations.

In the present clinical study about 76% of the patients were poor socio-economic class and remaining 24% were middle class which does not yield much statistical significance. But our hospital being a government hospital, which is serving most of the poor socio-economic status hence the percentage of poor socio-economic status are high. The diet pattern in this study showed 64% non-vegetarians and 36% were vegetarians which did not indicate any significance in relation to the disease.

In the present study of 50 cases of acute intestinal obstruction 40% of the cases are due to post operative adhesions who has undergone previous surgeries.

Table 12: Comparison of etiology with other studies

Cause	Souvik Adhikari	Jahangir	Arshad M. Malik	Cole GJ	Brooks and Buttler[45]	Playforth 1970[47]	Present Study
Adhesions	16%	49%	41%	10%	23%	54%	40%
Hernia	36%	34%	19%	35%	25%	23%	30%
Volvulus	6%	5%	4%	3%	1%	3%	4%
Tuberculosis	14%	1%	24%	3%	-	-	4%
Malignancy	17%	3%	2%	9%	5%	9%	14%
Intussusception	2%	6%	-	12%	18%	5%	6%
Mes.Ischaemia/ Miscellaneous	9%	2%	10%	-	-	6%	2%

In present study, postoperative adhesion is the commonest cause of intestinal obstruction, which is comparable with the other study groups Playforth et al. with 54% and Arshad Malik et al. with 41%. Although the incidence of obstructed/ strangulated hernia is more in the developing countries in this study group it is the second common aetiology for obstruction. It may be because the awareness of public, the availability of surgical facilities in the periphery for the hernia repair, the hernias are treated early.

Clinical features

The clinical feature of intestinal obstruction pain abdomen, vomiting distension of abdomen and constipation are not present in all cases. Pain abdomen was present in 88% of the cases in the present study, where as the vomiting was present in 78% of the cases. Whereas distension was present in 66% and constipation was present in 54% of the cases. The comparative table showing percentage of clinical features by various other study group are as follows.

Table 13: Comparison of clinical features with other studies

Study group	Pain abdomen	Vomiting	Distension	Constipation
Present study	88%	78%	66%	64%
Souvik Adhikari[42]	72%	91%	93%	82%
Jahangir-Sarwar Khan[43]	100%	92	97	97

In the present study, the clinical features of pain abdomen was 88%, vomiting was 78% which comparable with the other study group. Souvik Adhikari et al. and Jahangir Sarwar Khan et al. Only 66% of the patients in the present study group had distension of abdomen. It may be due to early approach to the hospital by patients in the present study.

The mass per abdomen on palpation is present in 24% of the total study move in Malignancy and ileocaecal tuberculosis. Visible peristalsis is present in 60% of the intestinal obstruction cases. The rectal examination did not reveal any abnormality except in four cases of intussusception (8%) and 2 cases of malignancy (4%) where in red current Jelly and rectal growth were the per rectal findings respectively.

Laboratory investigation

Among the total study population 30% of the cases were having Anaemia other wise the basic haematological investigation did not yield much statistical significance.

Radiology

The Erect abdomen X-ray helps us in the diagnosis of intestinal obstruction as well as in differentiating the small bowel with large bowel obstruction. Multiple our fluid level can be seen in small multiple intestinal obstruction where as only gas shadows seen in large bowel observation until the iceocaecal value is competent. Taneja et al. report shows 90% of cases with multiple air fluid level

and Savage et al. reports 95% cases with significant findings. In the present study of the 50 cases 60% of X-ray shows multiple air blood levels. Contrast study of barium enema may help to locate the obstruction in the colon but in our study contrast study was not done.

The surgical management for the present study group includes release of adhesions for postoperative adhesions 40%, resection of anastamosis for many of the cases of obstructed/strangulated hernia where the viability of the bowel was doubtful and also for ischaemic bowel 22%, release of constricting agents and herniorrhaphy was done in 18% of the obstructed/strangulated hernia cases. Derotation of volvulus and sigmoidopexy was done in 4% of the cases. Resection anastomosis and herniorraphy done in 8% of the cases. Reduction of intussusception in one case. Two cases were managed with Hartman's procedure and one case with transverse loop colostomy.

Complications

In the present study group out of 50 cases, complications like septicemia 5 cases, respiratory tract infection 2 cases, wound infection in two cases occurred. The complication of septicemia was more in the cases of malignancy and one case of mesenteric ischaemia case where in there was already sepsis at the time of admission, and for these cases bowel surgeries were done which were unprepared. Two cases one with obstructed inguinal hernia and one with the case of carcinoma rectum, the patients had prior comorbid conditions of COPD were suffered from respiratory tract infection.

Mortality

Frequency of mortality in our study is 14% i.e. 7 cases out of 50 cases. Among these 6 cases were due to malignancy and one due to mesenteric ischaemia.

Mortality that have occurred during various studies have been tabulated as follows.

Table 14: Morality rate in various studies

Studies	**Year**	**No. of cases studied**	**Mortality**
Present study	2009	50	14%
Souvik Adhikari[42]	2005	367	7.35%
Safian Matsu Moto[48]	1975	171	19%
Jahangir-Sarwar Khan[43]	2001	100	7%
Ramachandran CS[49]	1982	417	12.7%

The mortality rate in the present study is much comparable to Ramachandran CS et al. study but it is more when compared to Souvik Adhikari et al., Jahangir et al. studies.

Out of 7 cases died, 6 cases were due to malignancy. As the malignancy was more in the aged group and the unprepared bowel surgeries done to the patient led to septicemic condition and resulted in death. Two patients were chronic smoker who suffered respiratory tract infection and died. Hence most of the deaths were due to malignancy which played significant role in the outcome of the disease.

The mortality in intestinal obstruction is more in patients who develop strangulation and gangrene of the bowel, also who reached the hospital after 3 days.

With all these, the age of the patient, general condition of the patient, duration of symptoms. Operative procedures carries a high role in progress as well as the mortality.

Table 15: Comparison of mortality in relation to duration of symptoms with other studies

Sl. No.	Duration of symptoms before hospitalization	No. of cases	Death
1	1-2 days	18	2
2	3-4 days	17	2
3	> 5 days	15	3
Total		**50**	**7**

The table shows the time interval from the onset of symptoms to the hospitalization and the relation with complications as well as death. The prognosis of the patient is directly proportional to the duration of the disease symptom i.e. higher the duration higher the mortality.

CONCLUSION

- Acute intestinal obstruction remains an important surgical emergency in the surgical field.
- Success in the treatment of acute intestinal obstruction depends largely upon early diagnosis skilful management and treating the pathological effects of the obstruction just as much as the cause itself.
- Erect abdomen X-ray is valuable investigation in the diagnosis of acute intestinal obstruction.
- Postoperative adhesions are the common cause to produce intestinal obstruction. Clinical radiological and operative findings put together can diagnose the intestinal obstruction.

- Mortality is still significantly high in acute intestinal obstruction.

S.No	Name	Age	Sex	IP No	S E St	DIET	PA	VO	Di	CO	DOS	VIP	Tachycardia	Tenderness/ Rigidity/ Rebound tenderness	Previous scar	Mass PA	PR	Diagnosis	Blood	X ray abdomen erect	Management	Cured	Complications	Death
1	Srinivas	17	M	459	Mi	Mx	P	A	P	A	3	P	P	R	P	A	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	WI	-
2	Puttamma	20	F	519	L	V	P	P	P	A	5	A	A	RT	A	P	RCJ	Intussusception	WNL	ND	Reduction	C	-	-
3	Madaiah	66	M	1129	L	Mx	P	P	P	P	2	P	P	R	P	A	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	-	-
4	Susheel	23	m	2173	L	V	P	P	P	A	2	P	P	RT	P	A	N	AIO due to PO adhesions	WNL	ND	ROA	C	-	-
5	Izlum Rahman	55	M	2373	L	V	P	P	P	P	4	P	P	R	P	A	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	-	-
6	Tolasamma	70	F	2737	L	Mx	P	A	A	P	3	A	P	RT	A	P	RCT	Intussusception	WNL	ND	RA	C	-	-
7	Raghu	15	M	3009	Mi	V	P	P	P	A	4	P	P	R/T	P	A	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	-	-
8	Doddamma	75	F	3083	L	V	A	A	P	A	3	P	P	R	A	A	N	AIO due to ca sigmoid colon	WNL	MAFL	RA	-	SEP	D
9	Govindaswamy	71	M	4201	L	Mx	P	P	P	P	2	P	P	R/T	A	A	N	Str IH	AN	ND	RA&H	C	WI	-
10	Sundaramma	30	F	4842	L	Mx	P	P	A	A	3	A	P	RT	A	A	N	OB IH	WNL	ND	RA&H	C	-	-
11	Shivanna	72	M	5005	L	Mx	P	P	P	P	8	P	A	T	A	P	MASS PR	AIO due to ca rectum	AN	LBO	Hartman's procedure	-	RTI	D
12	Swamy	23	M	5086	L	Mx	P	P	P	A	1	P	P	R	P	P	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	-	-
13	Bhagyalakshmi	26	F	5680	Mi	Mx	A	P	P	P	2	P	A	T	A	P	N	AIO due to ICT	WNL	MAFL	RA	C	-	-
14	Basappa	85	M	6492	L	V	P	P	P	A	5	P	P	R/T	P	A	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	-	-
15	Shankaregowda	64	M	6604	Mi	Mx	P	P	A	A	3	A	P	T	A	A	N	OB IH	WNL	ND	RA	C	-	-
16	Devaraj	60	M	6990	L	Mx	A	A	P	A	2	A	P	T	A	A	N	OB IH	WNL	ND	ROA&H	C	-	-
17	Manjunath	25	M	7604	L	V	A	P	P	P	2	P	P	R/T	P	A	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	-	-
18	Puttathayamma	70	F	8860	Mi	V	P	P	P	A	3	A	P	R/T	A	A	RCJ	Sigmoid volvulus	WNL	ND	DVS	C	-	-
19	Munira Bai	50	F	9147	L	Mx	P	A	P	A	2	A	P	R/T	A	A	N	Str IH	WNL	ND	RA	C	-	-
20	Sudhakar	60	m	9246	L	Mx	P	P	A	P	3	P	A	R	A	A	N	OB IH	WNL	ND	ROA&H	C	-	-
21	Papanna	65	M	10621	L	Mx	P	P	P	P	5	A	P	T	A	A	N	AIO due to mesenteric ischaemia	HC	MAFL	RA	-	SEP	D
22	Malagaiah	45	M	l	L	V	P	A	P	P	3	A	P	R	A	A	N	Sigmoid volvulus	WNL	MAFL	DVS	C	-	-
23	Iqbal Pasha	40	M	11698	L	Mx	P	P	P	P	2	P	A	R	P	A	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	-	-
24	Nagraju	40	M	12665	Mi	V	P	P	P	A	3	A	P	R	A	A	N	OB IH	WNL	ND	ROA&H	C	-	-
25	Somanna	45	m	13115	L	Mx	P	P	P	A	2	P	P	R/T	A	A	N	Str IH	WNL	ND	RA	C	-	-

S.No	Name	Age	Sex	IP No	S E St	DIET	PA	VO	Di	CO	DOS	VIP	Tachycardia	Tenderness/ Rigidity/ Rebound tenderness	Previous scar	Mass PA	PR	Diagnosis	Blood	X ray abdomen erect	Management	Cured	Complications	Death
26	Poojaiah	60	M	14057	L	Mx	P	P	P	P	4	P	A	T	P	A	N	AIO due to PO adhesions	AN	MAFL	ROA	C	-	-
27	Raju	35	M	14117	Mi	Mx	P	P	P	P	1	P	P	RT	P	A	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	-	-
28	Kenchamma	69	F	14258	L	V	P	P	P	P	3	A	A	R/T	A	P	RCJ	Intussusception	WNL	-	RA	C	-	-
29	Manjulamma	40	F	14497	Mi	Mx	P	P	P	A	5	P	P	R/T	P	A	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	-	-
30	Puttaswamy	29	M	14610	L	V	P	P	P	P	4	P	A	T	P	A	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	-	-
31	Mahadeva	18	m	15458	L	Mx	P	P	P	P	3	P	P	T	A	A	N	OB IH	WNL	-	ROA&H	C	-	-
32	Lakshmi	25	F	14885	L	Mx	P	A	P	P	3	P	P	T	P	A	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	-	-
33	Nagaraju	35	M	16265	L	V	P	P	P	P	1	P	A	RT	P	A	N	OB IH	WNL	ND	ROA&H	C	-	-
34	Doddamallappa	65	m	17493	L	Mx	P	P	P	A	2	A	A	R	P	A	N	OB IH	WNL	ND	ROA&H	C	-	-
35	Naveen	18	m	19436	Mi	Mx	P	A	P	A	3	A	P	R/T	P	P	N	OB IH	WNL	ND	ROA&H	C	-	-
36	Madegowda	45	M	20239	L	Mx	A	A	P	P	3	P	A	T	A	P	N	AIO due to ca caecum	AN	MAFL	RA	-	RTI	D
37	Somasundari	38	F	20976	L	Mx	P	P	P	A	5	P	P	R/T	A	P	N	AIO due to ca ovary WIth sigmoid colon	AN	MAFL	TLC	-	SEP	D
38	Mahadevaiah	35	M	23274	L	V	P	P	P	P	3	P	P	T	P	A	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	-	-
39	Lingashetty	63	M	25375	L	V	A	P	P	P	3	A	P	R	A	A	MASS PR	AIO due to ca rectum	WNl	MAFL	Hartman's procedure	-	SEP	D
40	Mani	25	F	27583	L	Mx	P	P	P	P	2	A	P	R/T	P	A	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	-	-
41	Beeregowda	38	m	29319	Mi	Mx	P	P	P	A	4	A	P	T	P	A	N	OB IH	WNL	ND	ROA&H	C	-	-
42	Bellaiah	40	M	29681	L	Mx	P	P	P	P	3	P	A	R/T	A	A	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	-	-
43	Bora Venkataiah	55	M	29726	L	V	P	P	P	A	4	P	P	R/T	A	P	N	AIO due ca colon	WNL	MAFL	RA	-	SEP	D
44	Manchaiah	55	M	30161	Mi	Mx	P	P	P	A	1	A	P	RT	A	A	N	OB IH	WNL	ND	RA&H	C	-	-
45	Pradeep	20	m	30193	L	V	P	P	P	A	2	A	P	R	A	P	N	OB IH	WNL	ND	ROA&H	C	-	-
46	Indramma	55	F	30230	L	V	P	A	P	A	5	P	P	T	P	A	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	-	-
47	Nazeera Banu	40	F	30741	Mi	Mx	P	P	A	P	2	P	A	R/T	P	P	N	St IH	WNL	MAFL	RA&H	C	-	-
48	Shibrathulla Khan	52	M	30844	L	Mx	P	P	P	P	1	P	P	R	P	A	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	-	-
49	Chikkamuddaiah	60	M	31457	L	Mx	P	P	P	P	5	P	P	R/T	A	P	N	AIO due to ICT	WNL	MAFL	RA	C	-	-
50	Kamalamma	58	F	31649	L	Mx	P	A	P	P	3	P	P	R/T	A	A	N	AIO due to PO adhesions	WNL	MAFL	ROA	C	-	-